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EQUINE OSTEOMALACIA [1]

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TWENTY-FOUR PLATES AND SIXTEEN TEXT FIGURES

INTRODUCTION

The prevalence of osteomalacia among animals in the Philippine Islands, its enormous economic importance both to the United States Army and civilians, and the wealth of material found in the Philippines, prompted the Medical Department Research Board to undertake an extensive investigation of this condition. The United States Army in the Philippines has been hampered seriously by the prevalence of this disease among its animals and the situation is but little less serious with regard to other locally owned animals, particularly imported breeding stock.

A critical review of the literature pertaining to osteomalacia, of both animals and humans, indicates that considerable confusion exists regarding this condition. The efforts of the board in the present investigation have been directed toward practically all phases of the disease as it affects the equine species, and it is hoped that much of the existing confusion will be lessened as a result of this work.

Various terms such as osteomalacia, osteoporosis, osteitis fibrosa, big head, millers' disease, bran disease, cachexia osseus, halisteresis ostium, osteopsathyrosis, rarefying osteitis, osteitis deformans, osteitis fibrosystica, osteodystrophia deformans, mol-

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litis ossium, fragilitas ossium, etc., have been applied to this disorder by writers on the subject. Most of the terms used refer to a single predominating manifestation presented during the course of the disease and are not universal in their application. Of the many designations applied to this diseased condition in horses, the terms osteomalacia and osteoporosis are used most frequently. Some writers claim that osteomalacia and osteoporosis of equines are separate and distinct entities, others say that they are closely related conditions, while still others state that the conditions are identical. Mohler(1) claims that osteoporosis is a complete entity entirely independent of osteomalacia, citing the fact that osteomalacia occurs in range cattle in the southwestern part of the United States while osteoporosis in horses on the same range has not been noted. Further, he states that osteomalacia of cattle responds to treatment while osteoporosis of horses does not and that osteomalacia occurs in animals that graze on worn-out soil deficient in lime salts while osteoporosis may occur in animals that graze on rich fertile soil. Robertson(2) states that osteoporosis and osteomalacia are totally different diseases; different in cause, nature, treatment, and post-mortem lesions. He claims that the cause of osteomalacia is defective nutrition while osteoporosis is not induced in this manner. Further, that osteoporosis attacks only the Equidæ while osteomalacia may affect all domestic species. Forbes(3) states that osteoporosis appears to be distinct from osteomalacia in that the former disease does not respond to treatment with calcium phosphate and its etiology is unknown. This writer quotes D. Hutcheon, chief veterinary surgeon, Cape of Good Hope, who attempts to differentiate the two conditions on a pathological basis. Marek(4) claims that in osteoporosis there is an absence of osteoid tissue and that the two conditions are differentiated by the pathogenesis and the histological picture. Further, he states that osteoporosis may occur concurrently with osteomalacia. As opposed to these views, many investigators say that the conditions are identical or are related so closely that they should be considered as being the same disease. Friedberger and Frohner(5) in discussing osteoporosis state that the condition is related to osteomalacia and should not be considered a distinct disease. Winslow(6) recognizes the confusion that exists in designating the disease and prefers the term osteomalacia, stating that this term properly includes the various pathological conditions to which numerous names have been given. Law(7) states that osteo-

porosis is a form of osteomalacia occurring in horses. White(8) in his text on veterinary medicine discusses the two conditions under the same heading. Bordeaux(9) considers osteoporosis and osteomalacia of equines identical and prefers the latter term. Sturgess,(10) in a recent investigation of the condition in Ceylon, states that the term osteoporosis is not an exact description of the pathological changes occurring in the disease, but retains the term as it is the name with which horse owners in Ceylon are familiar. Miller(11) states that certain authorities claim that osteoporosis is associated with osteomalacia and may be a form of the same disease.

That the specific pathological process recognized as osteoporosis, abnormal porosity or rarefaction of bone, occurs in the condition under investigation is admitted by practically all investigators. Certain other pathological processes are usually present so that the complete micro- and macropathological picture is more than a simple osteoporosis. The writers, therefore, have used the term osteomalacia, as it includes the several pathological conditions present during the course of the disease and is based upon certain specific histological findings.

DEFINITION, HISTORY, AND OCCURRENCE

Definition.—Osteomalacia of animals is a disturbance of the mineral metabolism of the bones, possibly inflammatory in nature, wherein preëxisting bone is removed and replaced in part by osteoid tissue. Osteoporosis or rarefaction occurs concurrently with the condition. The disease is usually chronic but may be acute or subacute and usually involves all of the bones of the body.

History and occurrence.—Osteomalacia is one of the oldest diseases known and, according to Friedberger and Frohner,(5) was described accurately by Vegetius in the fourth century. Extensive outbreaks of the disease among animals are reported from Germany as early as 1778.(12) According to McCrudden,(13) Chossot (1842) was the first to produce artificial osteomalacia, which he accomplished by feeding pigeons a diet poor in calcium salts.

The disease has been reported in the following countries, some of which have had extensive outbreaks: Belgium, Germany, France, and Austria;(12) England, Scotland, Russia, and South Africa;(2) Hawaii;(14) parts of the United States and the Philippine Islands;(4) the Sandwich Islands;(15) the Transvaal;(16) New South Wales and India;(17) Uruguay;(18) and

the Belgian Congo.(19) Per Tuf(20) reports that in Norway, where it has now lost much of its importance, severe outbreaks affecting cattle and horses occurred in 1860, in 1869 (over 2,000 cases), in 1876–77 (over 3,000 cases) and extensive outbreaks in 1904, 1905, 1911, and 1912. An epizoötic of the disease occurred in Switzerland in 1919.(21) Sturgess(10) states that in Colombo, Ceylon, at least 10 per cent of the horses are affected in varying degrees. The condition occurs in Brazil where it is known as "cara inchada."(22) It occurs enzoötically in certain regions of the United States, especially in New Jersey and the Mississippi Valley.(7) It is common in certain parts of the Philippine Archipelago.

While cattle, particularly dairy cows, are affected most frequently, horses, mules, asses, goats, dogs, sheep, and pigs are susceptible to the disease. Blair and Brooks(23) state that it is common among primates at zoölogical gardens, where it is known as "cage paralysis." Fox,(24) White,(25) and others report its presence among captive wild animals and birds. Law(7) reports that it is particularly common among Shetland ponies, while asses and mules habitually escape the disease.

Osteomalacia appears to be enzoötic in character. Many severe outbreaks are reported as having appeared after prolonged droughts. Some writers attach particular importance to the mineral nature of the soil, reporting either calcium or phosphate deficiency in affected areas. Mohler,(1) however, states that equine osteoporosis is found in limestone sections of the United States. In Ceylon, Sturgess(10) reports that there is very little difference in the incidence from year to year and that the disease is not seasonal.

Osteomalacia of humans has been reported from practically all civilized countries, and many extensive investigations have been carried out. A critical review and an able discussion of the literature on the subject have been published recently by Hess.(26)

SIMILAR DISEASES

Similar diseases.—Diseases of disturbed mineral metabolism have attracted considerable interest for many years. Due to recent advances in biological chemistry, in the knowledge regarding the physiological action of several of the glands with internal secretions, to the discovery of the vitamins, to the ability to produce several diseases of this character in experimental animals, and to the indicated significance in nutrition

of several of the chemical elements, enormous quantities of interesting data have been published regarding mineral metabolism and diseases concerned therewith. A full discussion of all conditions related to osteomalacia is not contemplated, therefore, in the present paper. While the recent advances in the knowledge of mineral metabolism of animals have been exceedingly rapid and fruitful, the complex metabolic processes involved are not understood fully and it is impossible, therefore, to correlate properly the anatomical and physiological differences between the various species.

Rickets.—Until comparatively recent years considerable controversy existed concerning the relationship of rickets and osteomalacia. Hess(26) and others state that Virchow in 1853 advanced the theory that rickets and osteomalacia are different diseases, stating that in the latter disease there is absorption while in the former nothing essential is absorbed. Many of the early investigators believed in Virchow's theory. In 1885, Pommer demonstrated that bone is not a fixed tissue but is subject to changes throughout life. Pommer also claimed that in both rickets and osteomalacia the lessions are due, not to an increased absorption but to a decreased calcification.(26) Von Recklinghausen, as quoted by Hess, believed that both rickets and osteomalacia were associated with dissolution of bone, halisteresis, while Schmorl and Looser maintain that these diseases are due to failure of calcification and not to halisteresis. Wells(27) states that in rickets there is failure of the osteoid tissue to calcify while in osteomalacia absorption of calcified tissue takes place. Law(7) believes that in rickets the young bone is attacked at the seat of growth and under the periosteum while in osteoporosis (osteomalacia of the horse) the process attacks mature bone in its interior, producing a thinning of the walls of its vascular canals, cancellar cavities, and increasing the fibrocellular contents. Further, he states that the bony enlargements in rickets are due to deposition on the bone surface, while in osteomalacia expansion takes place from within. More-recent observers, however, are agreed that in rickets and osteomalacia the essential abnormality is a deficient calcification of the osteoid tissue and that the latter condition is really a manifestation of rickets in adult life. Steenbock and Hart(28) state that rickets, osteoporosis, and osteomalacia are all characterized by impoverishment of the bones in inorganic substances. Hess(26) believes that there is no essential difference

between rickets and osteomalacia and that the difference in the pathological lesion between the two conditions is quantitative. Friedberger and Frohner(5) state that the two diseases are related and the differences in the pathological changes of the bone are due to age differences. Marek(4) agrees with Friedberger and Frohner and states that the difference is caused solely by the age of the diseased animal; he states further, that both diseases are identical in their nature, and the condition may be present in the same animal before it reaches maturity. Hutyra and Marek(12) believe that, while rickets and osteomalacia have much in common and that their anatomical and histological differences are due largely to the differences in ages of affected animals, the two conditions should be separated clinically and anatomically because of the age differences and the occasional etiological differences. Van Saceghem(19) contends that rickets, osteomalacia, and osteoporosis arise from identical causes and suggests the term "osseous cachexia" for the three diseases.

Equine osteoporosis.—The relationship of the so-called equine osteoporosis to osteomalacia has been considered in the introduction to this report. It is believed that the term osteoporosis should be used exclusively as a descriptive term of a specific pathological process (porosity or rarefaction of bone) and not as a disease entity.

Senile osteoporosis.—Senile osteoporosis, sometimes called senile osteomalacia, is a normal physiological process seen in the aged and causes atrophy and increased fragility of the bones without marked deformity. Senile osteoporosis differs chiefly in that no new osteoid tissue is formed.(27) This process may lead to such rarefaction that fractures are common.(29)

Styfsiekte.—This disease occurs among cattle of South Africa and is now recognized as a disease of disturbed mineral metabolism closely related to osteomalacia. Theiler, Green, and du Toit,(30) Malan, Green, and du Toit(31) and others have conducted extensive investigations of this condition. Hunter(32) in reviewing work on calcium and phosphorus metabolism states that styfsiekte is rickets, or osteomalacia, of a low phosphorus type.

Kennel lameness.—Brimhall and Hardenbergh(33) and Hardenbergh(34) report an outbreak of "rheumatism," or "kennel lameness," among mature dogs in which there was softening and deforming of certain bone structures approximating osteomala-

cia. In the latter report the writer considers the disease to be a type of osteomalacia.

Other diseases of bones.—The relationship of various exostoses commonly seen in horses as spavins, ringbones, and other bone diseases is unknown. Law(7) states that W. L. Williams noticed that for years after outbreaks of equine osteomalacia on two farms in central Illinois there was an unusual prevalence of spavins, ringbones, and other bone diseases. In connection with work on osteomalacia and rickets at the New York State Veterinary College(35) it was found that spavins and ringbones responded to treatment for osteomalacia and rickets.

It has been noted in the present investigation that exostoses of various types as well as numerous skin affections are associated commonly with osteomalacia of Army animals in the Philippine Islands.

ECONOMIC IMPORTANCE

Economic importance.—That such a disease of animals as osteomalacia is of tremendous economic importance needs no proof. The value of animals lost or incapacitated for work during long periods as a result of the disease cannot be estimated, but the loss is high. The United States Army in the Philippine Islands has been hampered seriously by the prevalence of this disease among its animals and the condition has led to a recommendation by at least one high Army commander that all units be motorized unless animals are indispensable. The situation is but little less serious with regard to native animals and animals imported by the Government of the Philippine Islands in its attempt to improve the quality of native stock by the use of pure-bred stallions. At the Trinidad Stock Farm of the Insular Government the condition was so serious that abandonment of the farm as a breeding station for horses was necessary.

The many reports of the presence of osteomalacia among animals of other countries prove it is a disease of prime importance in several of them.

PRESENT INVESTIGATION

GENERAL

Animals.—The present study of equine osteomalacia was begun in May, 1930, continued uninterruptedly for a period of approximately eighteen months, and was confined almost exclu-

sively to United States Army animals in the Philippine Islands. During the period of investigation the Army animal strength of the Philippines was about 3,000 head, approximately 40 per cent of which were horses and 60 per cent mules. All of these animals, with the exception of a few that were foaled here from Army mares, were imported from the United States at various times during the past twenty years. Their source in the United States was largely the midwestern and southwestern sections of the country. From time to time as replacements are required, animals are shipped by Army transport to Manila.

Army animals in the Philippines are maintained at several Army posts, the largest group being stationed at Fort Stotsenburg, Pampanga (approximately 2,000 in number) and Fort William McKinley, Rizal (approximately 700). Smaller groups of from 20 to 100 are stationed at the Post of Manila (Manila), Camp Nichols (Rizal), Fort Mills (Corregidor), Camp John Hay (Mountain), and Zamboanga (Mindanao).

Animal management.—At large posts, namely Fort Stotsenburg, Fort William McKinley, and the Post of Manila, a veterinary service, including resident veterinary officers, supervises the animal management and provides care and treatment for all sick and wounded animals. The veterinary service at the smaller posts is provided by frequent visits of a veterinary officer. The system of veterinary service at the larger stations provides for frequent physical examination of all animals and a complete system of records of all animals unable to do full military duty. A definite means of identification of individual animals is provided for by the Preston Brand System, in which each animal is indelibly branded on the left side of the neck at time of entry into service. With this system of veterinary service, together with the recording of all disabilities of individual, definitely identified animals throughout their Army service, it has been possible to obtain valuable data.

The ages of Army animals in the Philippine Islands range from 4 years to approximately 30 years, the majority falling in the age group 10 to 16 years. Replacement animals coming to the Philippines vary in age from 4 to 7 years. It is estimated that the military life of a horse is about twelve years and that of a mule about fourteen years.

Forage and water.—In the early days of occupation in the Philippines, some of the animals were rationed entirely on native forage because sufficient American feed was not available.

The majority were fed imported forage. The ration for native ponies used by the Army in 1899 consisted of rough rice, tikitiki, molasses, and green grass. About 1907, with the idea of utilizing native forage as an economic substitute for a portion of the imported feed for American horses, and in order to make the Philippine Islands more self-sustaining, investigative work was started by Army authorities in coöperation with the Bureau of Agriculture of the Insular Government on the possibility of substituting native for imported feeds. Tests of various crops suitable for hay and green forage were conducted at different points throughout the Islands, but no definite conclusions were drawn.

In 1910, the Secretary of War appointed a board, members of which were civilians and military officers interested in the forage question in the Philippine Islands. The United States Department of Agriculture detailed a special representative to coöperate with this board. During the period September, 1910, to May, 1911, this board conducted about twenty-five feeding tests, using native and various combinations of native and American forage. Several rations were reported as being satisfactory, but the amount of suitable native products was insufficient to meet the demands of the Army. This board reported that native cracked corn and native palay (rough rice) could be fed in lieu of a portion of the imported oats and that native green grass could be substituted for a portion of the imported hay allowance.

For a year or two after the tests conducted by the forage board, attempts to obtain local hay met with some success at the different posts. At most places, however, there was an insufficient quantity available and necessary steps were not taken to increase the production. As a consequence the roughage component of the ration soon reverted to American hay. The feeding of approximately 11 pounds of native green grass in lieu of one-third of the hay allowance was in effect at this time.

The development of the present forage ration was started in 1922 when a board of Army officers was appointed to investigate the suitability of copra meal, a by-product in the coconut industry, as an article of food for animals. At this time the Army ration consisted of American oats, American grain hay, and native green grass. After numerous feeding experiments the board reported that a ration composed of one-

third American oats, one-third copra meal, and one-third binilid or palay was considered satisfactory and this was adopted for the concentrate portion of the ration for Army animals.

In 1923 another forage board of Army officers was appointed and a careful investigation was made of the local forage situation. This board recommended a continuation of the grain ration of one-third oats, one-third copra meal, and one-third palay, and, further, that native rice hay be substituted for one-half of the American-hay allowance. Some time elapsed after the adoption of this ration before production of local forage permitted its general use. In the early experiments with rice hay, considerable difficulty was experienced in obtaining a product that was harvested at the proper stage of maturity and was well cured. This difficulty has been overcome to a great extent by requiring that the hay be cut while in the "milk stage" and the product, as now used, offers a fairly satisfactory substitute for a portion of the hay allowance.

With minor changes only, the following ration and allowance were in effect from 1923 to June, 1931:

TABLE 1.—*Forage for public animals.*

Animal.	Type.	American grain hay.	Native rice hay.	Native green forage.	Oats.	Native crushed palay.	Native copra meal.	Total. grain.
Horse	1,150 lbs. and over	5	5	12½	4	4	4	12
Do	900 to 1,150 lbs.	4½	4½	12½	3	3	3	9
Do	Under 900 lbs.	4½	4½	12½	2⅓	2⅓	2⅓	7
Mule	Draft	4½	4½	12½	2⅔	2⅔	2⅔	8
Do	Pack and saddle	4½	4½	12½	2⅓	2⅓	2⅓	7

The allowances in Table 1 represent the ration for animals doing garrison duty, practice marches, maneuvers, or field service of three days or less duration. The daily allowance of salt is 0.8 ounces per animal. For idle animals or those doing a small amount of light work, the concentrates of the ration are reduced 50 per cent. Grazing of animals is required whenever practicable. All forage is subjected to a critical inspection for sanitary condition and quality at the time of purchase.

During the fiscal year 1925 when the average daily strength was 4,000 animals the estimated savings that resulted from the partial substitution of native products for imported forage, was

approximately 75,000 dollars. Due to the marked advance in the price of American farm products during the fiscal year 1926, the estimated savings by employing partial substitution of native products was approximately 200,000 dollars. Based upon the prices of forage and the average animal strength for the fiscal year 1930, a saving of approximately 87,700 dollars resulted from the utilization of native products.

In view of the savings effected by the use of native products, and the economic and informatory benefit derived by the civilian agricultural and livestock interests, one of the chief factors considered in the present investigation was the use of as large a proportion of Philippine products as was consistent with favorable results.

Drinking water for animals is secured from artesian wells at all posts except Fort Stotsenburg and Post of Manila. At the former post it is secured from Bamban River and Tipley Creek, while the city water supply of Manila is utilized at the latter post.

The routine care of the Army animals requires thorough daily grooming, daily exercise, grazing whenever possible, and daily veterinary inspection of the animals and the sanitary conditions under which they are kept. All animals are stabled in open-type stables.

INCIDENCE

General.—Since osteomalacia is a disease affecting the skeleton and, in the Equidæ involves usually the organs of locomotion, it is included in Group X, "Diseases of the Bones and of the Organs of Locomotion," of the International Classification of Diseases. While this group includes many conditions unrelated to osteomalacia, it is of interest to compare the incidence among Army animals in the United States with Army animals in the Philippine Islands. Both groups of animals are maintained under similar conditions with the exception of climate, feed, and water. The geographical source, the breed, the type, ages, animal management under which maintained, etc., are practically the same for each group.

Statistical data from Reports of the Surgeon General, United States Army, showing the admission rates for diseases of the bones and of the organs of locomotion among Army animals in the United States and in the Philippines for the years 1923 to 1929, inclusive, are given in Table 2 and fig. 1.

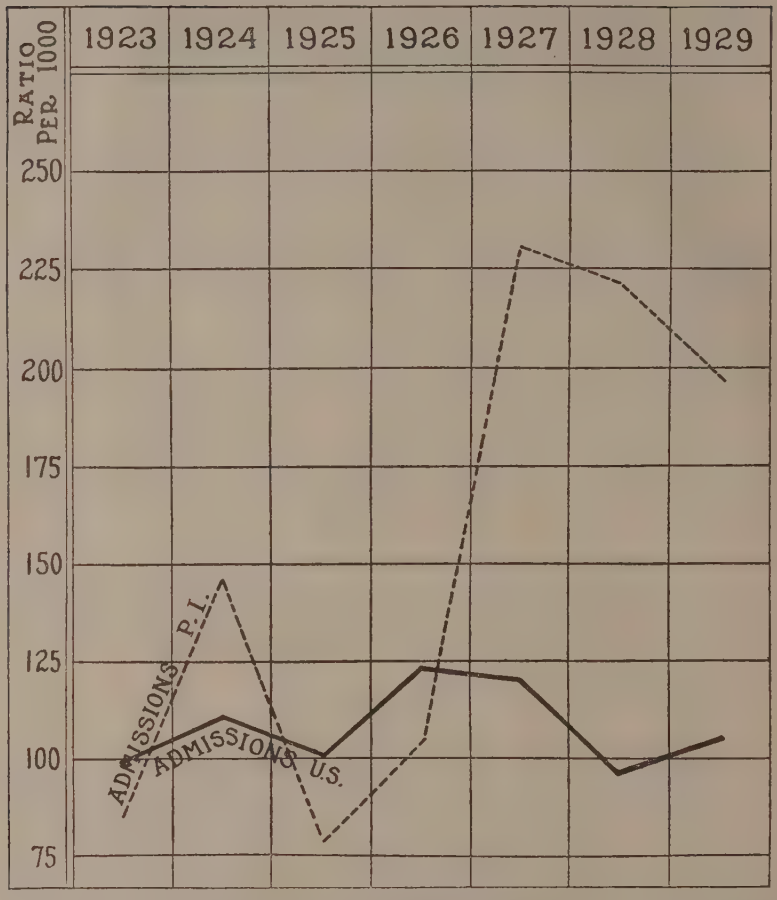

FIG. 1. Admission ratios: "Diseases of Bones and of the Organs of Locomotion."

TABLE 2.—*Admission ratios Group X: "Diseases of Bones and of the Organs of Locomotion."*

Year.	Total admission ratio per 1,000.	
	U. S.	P. I.
1923 [a]	99.20	84.28
1924 [a]	109.82	146.55
1925 [a]	100.84	79.15
1926 [a]	123.89	105.50
1927 [a]	120.45	231.13
1928 [a]	96.93	220.00
1929 [a]	106.78	192.27

[a] Abstracted from Surgeon General's Report for following year.

It will be noted that a marked increase of the admissions for diseases of this group occurred during recent years among animals in the Philippines and that the ratio is decidedly higher for Army animals in the Philippines than for like groups of animals in the United States.

Osteomalacia is an insidious disease, obscure in its initial stage, usually slow in its development, and intimately associated with other conditions that result in disturbed locomotion. While several cases of osteomalacia among Army animals in the Philippines had been reported prior to July, 1930, its importance was not recognized until after this date. The complex syndrome accompanying osteomalacia is confused easily with other conditions and a diagnosis of arthritis, obscure ringbone, sprain of joints, undetermined lameness, etc., probably was made in many instances. As previously noted in the discussion on similar diseases, osteomalacia may be related to ringbone and arthritis chronica deformans tarsi.

The incidence of diseased conditions likely to be confused with, and possibly related to, osteomalacia among Army animals in the United States and in the Philippines for the years 1923 to 1929, inclusive, are shown in Table 3 and fig. 2. These data are compiled from Reports of the Surgeon General of the Army.

TABLE 3.—*Admission rates for conditions indicated. Admission ratio per 1,000.*

Disease.	1923		1924		1925		1926	
	U. S.	P. I.	U. S.	P. I.	U. S.	P. I.	U. S.	P. I.
Arthritis	9.26	12.42	14.50	18.77	14.33	9.10	18.55	6.54
Arthritis chronica deformans tarsi	8.37	6.39	8.00	19.66	6.33	15.25	10.59	8.89
Ringbone	6.97	6.45	8.85	27.49	6.06	8.23	10.47	15.17
Sprain of joints	12.76	.....	13.42	24.22	15.18	48.50	17.77	52.80
Total	37.36	25.26	44.77	90.14	41.90	81.08	57.38	83.40

Disease.	1927		1928		1929	
	U. S.	P. I.	U. S.	P. I.	U. S.	P. I.
Arthritis	16.95	22.77	11.92	25.64	14.14	13.70
Arthritis chronica deformans tarsi	9.09	20.30	9.32	12.53	12.30	31.91
Ringbone	10.99	24.54	9.09	26.60	11.71	22.44
Sprain of joints	16.02	44.41	13.39	31.41	13.56	43.62
Total	53.05	112.02	43.72	96.18	51.71	111.67

It will be noted that the admissions for arthritis, arthritis chronica deformans tarsi (spavin), ringbone, and "sprain of joints" were considerably greater for the group of Army animals in the Philippines during the period 1924 to 1929, inclusive, than for the group in the United States during the same period.

With an admission rate of 34.20 cases per 1,000 for these diseases among animals in the Philippines for 1923, this rate increased threefold for 1924, or 106.30 per 1,000, and remained at a high level during the period 1925 to 1929, inclusive. The rates for 1930 are not available at this time.

It should be noted that the sudden increase in the diseases mentioned occurred in the year following the change of ration (1923).

Due to the fact that the largest group of Army animals (approximately 2,000) in the Philippines is stationed at Fort Stotsenburg, Pampanga, and that the highest incidence of the disease occurred at this station, a detailed study of the condition was made at this post. Table 4 shows the animal strength of the two largest Army posts in the Philippines and indicates the rates for osteomalacia at these stations from July 1, 1930, to July 1, 1931.

TABLE 4.—*Mean strength and osteomalacia rates; July 1, 1930, to July 1, 1931.*

Station.	Mean animal strength.	Total animals affected.	Annual ratio per 1,000.
Fort William McKinley	684.01	94	137.4
Fort Stotsenburg	2,139.55	340	158.9

The veterinary records on file at Fort Stotsenburg furnish a complete history of the animal conditions at this post from 1922 to date. All animals at the post, with the exception of a small group of horses owned by officers, are definitely identified by means of the Preston Brand System, which was adopted in 1924. The Preston brand number is entered on all records pertaining to the animal, thus furnishing a complete veterinary history of each individual since June, 1924. An analysis was made of all records from January 1, 1925, to October 1, 1931. During the period June, 1922, to January, 1925, one case of osteomalacia (diagnosed as osteoporosis) was recorded. This occurred in 1924.

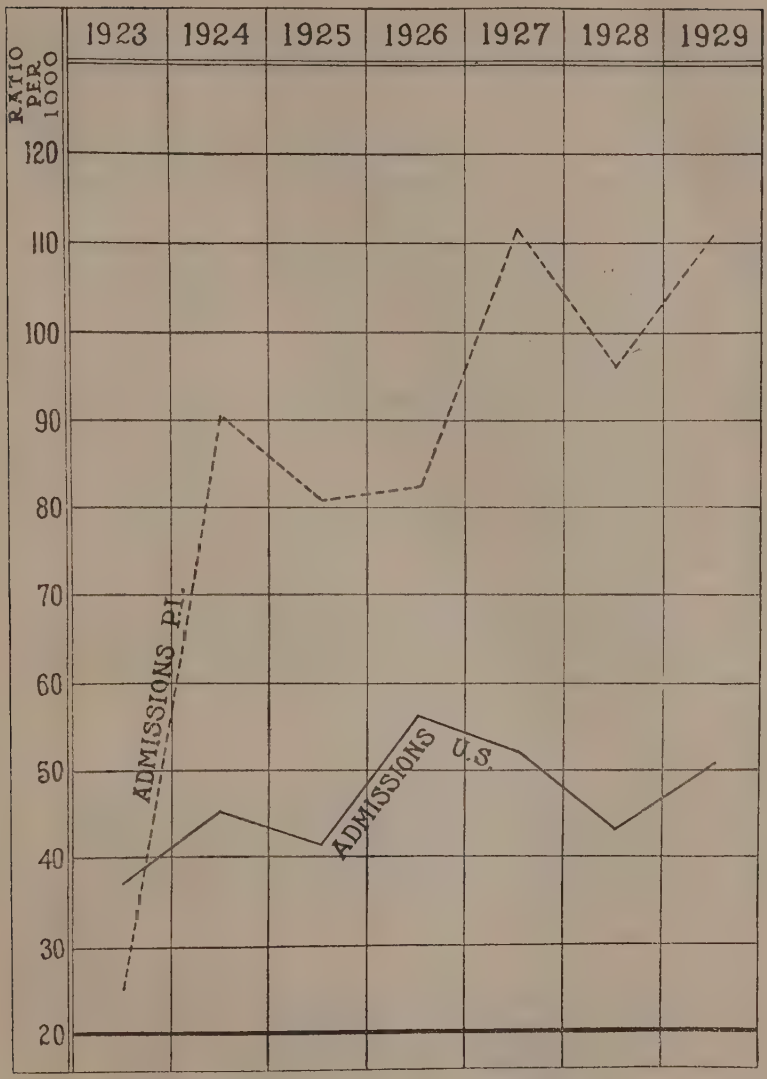

FIG. 2. Admission rates for conditions indicated in Table 3.

Table 5 shows that from January 1, 1925, to October 1, 1931, a total of 366 cases of osteomalacia were recognized. Of these cases, 340, or approximately 93 per cent, were diagnosed during the period July 1, 1930, to July 1, 1931.

TABLE 5.—*Showing in detail the cases of osteomalacia at Fort Stotsenburg from January 1, 1925, to October 1, 1931.*[a]

Year.	Period.	Species.	Status.		Sex.		Type.			Age.									
			Private mount.	Public animal.	Gelding.	Mare.	Riding.	Draft.	Pack.	4 years.	5 years.	6 years.	7 years.	8 years.	9 years.	10 years.	11 years.	12 years.	13 years.
1928	Entire year	Horse		1	1		1											1	
1929	do	do		3	1	2	3								1		1	1	
1930	January, February, March																		
	April, May, June	Horse	1	2	3		3					1					1		
	July, August, September	Horse		14	10	4	14					1		1	2	2	3	3	2
		Mule		7	4	3		3	4						1	4		1	
	October, November, December	Horse	4	62	52	14	65		1	1	1	1	1	2	10	10	14	13	6
		Mule		12	7	5		3	9		1		3		1		1	3	1
	Entire year	Horse	5	78	65	18	82		1	1	1	3	1	3	12	12	18	16	8
		Mule		19	11	8		6	13		1		3		2	4	1	4	1
		Total	5	97	76	26	82	6	14	1	2	3	4	3	14	16	19	20	9
1931	January, February, March	Horse	4	107	86	25	108	3			2	4	4	7	8	10	14	12	15
		Mule		40	23	17		15	25		1	1	5		3	3	2	5	2
	April, May, June	Horse		57	43	14	54	1	2		2	1	2	3	4	3	6	10	7
		Mule		33	23	10		7	26			3	1	2	3	2	6	2	5
	July, August, September	Horse	1	7	7	1	8							1	1				1
		Mule		11	5	6		5	6			1	1	1	2		1	1	
	January 1 to October 1	Horse	5	171	136	40	170	4	2		4	5	7	11	12	13	21	22	23
		Mule		84	51	33		27	57		1	5	7	3	8	5	9	8	7
		Total	5	255	187	73	170	31	59		5	10	14	14	20	18	30	30	30
Cases January 1, 1925, to October 1, 1931[a]		Horses	10	253	203	60	256	4	3	1	5	8	8	14	25	25	40	40	31
		Mules		103	62	41		33	70		2	5	10	3	10	9	10	12	8
		Total cases	10	356	265	101	256	37	73	1	7	13	18	17	35	34	50	52	39

Year.	Period.	Species.	Age.												Disposition.				Total cases.
			14 years.	15 years.	16 years.	17 years.	18 years.	19 years.	20 years.	21 years.	22 years.	23 years.	25 years.	25 years and over.	Destroyed.	Died concurrent condition.	Duty.	Remaining under treatment.	
1928	Entire year	Horse													1				1
1929	do	do													3				3
1930	January, February, March																		0
	April, May, June	Horse		1											3				3
	July, August, September	Horse													13		1		14
		Mule		1											6		1		7
	October, November, December	Horse	4		1				1		1				55		11		66
		Mule										1		1	11		1		12
	Entire year	Horse	4	1	1				1		1				71		12		83
		Mule		1								1		1	17		2		19
		Total	4	2	1				1		1	1		1	88		14		102
1931	January, February, March	Horse	3	2	2		1	5	3	8	1	5	4	1	72		38	1	111
		Mule	1	1	1	1		4		2	2	2	2	2	34		6		40
	April, May, June	Horse	6	2				2	1	4	2		2		16	1	35	5	57
		Mule	1						1	1		1	4	1	5		22	6	33
	July, August, September	Horse		1			1	2							3		5		8
		Mule			1	1				2					2		6	3	11
	January 1 to October 1	Horse	9	5	2		2	9	4	12	3	5	6	1	91	1	78	6	176
		Mule	2	1	2	2		4	1	5	2	3	6	3	41		34	9	84
		Total	11	6	4	2	2	13	5	17	5	8	12	4	132	1	112	15	260
Cases January 1, 1925, to October 1, 1931[a]		Horses	13	6	3		2	9	5	12	4	5	6	1	166	1	90	6	263
		Mules	2	2	2	2		4	1	5	2	4	6	4	58		36	9	103
		Total cases	15	8	5	2	2	13	6	17	6	9	12	5	224	1	126	15	366

[a] No cases recorded from January 1, 1925, to July 16, 1928.

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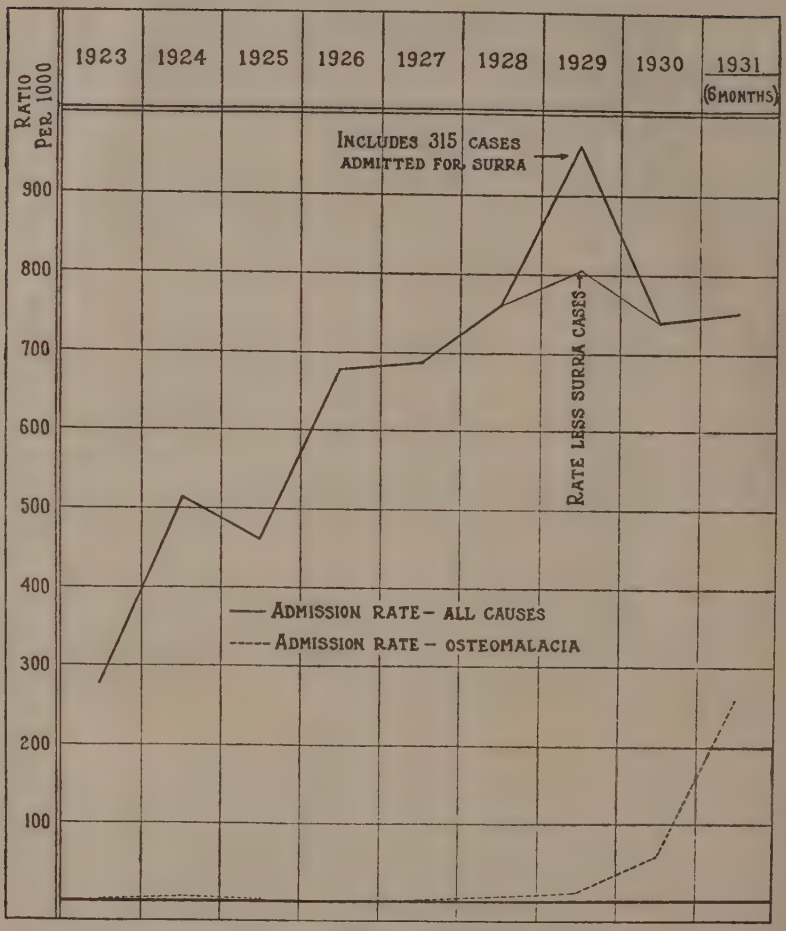

FIG. 3. Comparison of osteomalacia admission rates with total admission rates.

Table 6 shows the number of cases of the disease by year and the annual ratio per 1,000.

A comparison of the incidence of osteomalacia with all causes of admission to sick report is shown in Table 7 and fig. 3.

It will be noted, as shown in Table 7 and fig. 3, that a decided increase in the admission rate for all causes occurred in 1924, one year following the change of ration. While this rate decreased somewhat in 1925, it remained at a uniformly high level

from 1926 to July 1, 1931, with the exception of a sharp increase of total admissions in 1929, which was due to an epizoötic of surra, when 315 cases of this disease were admitted. The admission rate for osteomalacia was of little significance until 1930 when a marked increase occurred. It should be noted, as shown in fig. 3, that a decided increase in admission for osteomalacia occurred in 1930 and 1931. This increase did not affect the total admission rate. That the latter rate is affected markedly by admission of approximately 300 cases is indicated by the increased rate for 1929 when the surra outbreak occurred in the command. In 1931, during the first six months, 285 admissions for osteomalacia were recorded, a ratio of 570 cases per year. This large number of admissions did not affect the total admission rate. For the above reasons, it is believed that osteomalacia was much more prevalent in the command prior to 1930 than the records indicate.

The incidence of osteomalacia as compared with total admissions, for the period July 1, 1930, to July 1, 1931, is shown in fig. 4.

During the period indicated in fig. 4, 1,666 animals were admitted to sick report. Of this total 395, or 23.7 per cent, were admitted for osteomalacia.

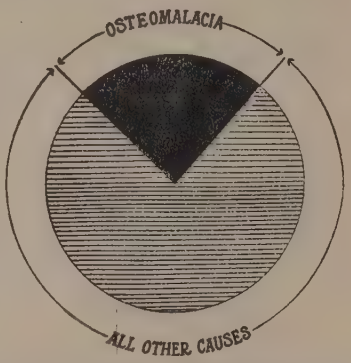

FIG. 4. Comparison of admissions for osteomalacia and admissions for all other causes.

Status.—Referring to Table 5, it will be noted that Army animals are divided into two groups; namely, private mounts and public animals. The group of private mounts includes approximately 86 horses owned by officers and used by them in the Army service; the group of public animals is composed of Government-owned horses and mules used by the Army. Private mounts are animals of superior type, conformation, and quality, and are better bred usually than are the horses of the public-animal group. The conditions under which the two groups of horses are maintained differ somewhat. The feed of the former

TABLE 6.—*Osteomalacia at Fort Stotsenburg, January 1, 1925, to October 1, 1931.*

Year.	Animals affected.	Annual ratio per 1,000.
1925	0	
1926	0	
1927	0	
1928	1	0.46
1929	3	1.55
1930	102	50.89
1931 [a]	260	171.22
Total	366	

[a] Nine months.

TABLE 7.—*Admissions and rates. Comparison of osteomalacia with all causes.*

Year.	Mean strength.	Admissions.		Annual admission ratio per 1,000.	
		All causes.	Osteomalacia.	All causes.	Osteomalacia.
1923	2,192	619		277.8	
1924	2,112	1,087	1	514.7	0.5
1925	2,157	989		458.5	0
1926	2,335	1,586		679.2	0
1927	2,308	1,579		684.1	0
1928	2,176	1,653	1	759.7	0.5
1929	1,994	[a] 1,916 [b] 1,601	3	[a] 960.9 [b] 802.9	1.5
1930	2,004	1,495	114	746.0	56.9
1931 [c]	2,117	797	285	752.9	269.2

[a] Total admissions and total admission rate.
[b] Admissions and rate less 315 cases of surra, which occurred during 1929.
[c] Six months.

group consists usually of American oats and American hay with an occasional addition of wheat bran, while the latter group is fed the prescribed Army ration. It will be noted in Table 5 that ten cases of osteomalacia occurred in animals of the private-mount group during 1930 and the first nine months of 1931, while 249 cases occurred in horses of the public-animal group. Based on the mean animal strength of these two groups (86 private and 797 public) 11.6 per cent of the private mounts and 31.2 per cent of horses belonging to the public group developed osteomalacia.

No definite conclusions are drawn with relation to the difference in incidence of osteomalacia between these two groups for the following reasons: (1) The ration fed to private mounts varies somewhat with the individual owner. Details regarding the ration for this group were unobtainable for the period noted. (2) The length of service in the Philippines for animals of the private-mount group is much shorter usually than is the case in the public-animal group. The tour of service for private mounts corresponds generally to that of the owners (two to three years). The public animals in the Philippines rarely are returned to the United States.

Species.—As already noted 366 cases of osteomalacia were recorded during the period January 1, 1925, to October 1, 1931; 340 cases, or approximately 93 per cent, were diagnosed during the period July 1, 1930, to July 1, 1931. It is believed that a more accurate picture of the incidence of the disease among horses, as compared with mules, is afforded by consideration of the cases occurring during the latter period, as the full significance was recognized during this period only.

Of the 340 animals affected during the period July 1, 1930, to July 1, 1931, 251 were horses and 89 mules. The incidence rates, based on the mean animal strength of the command during the period noted, are shown in Table 8.

TABLE 8.—*Species incidence.*

Species.	Animals affected.	Mean animal strength.	Annual ratio per 1,000.
Mule	89	1,204	73.9
Horse	251	936	268.3
Total	340	2,140	158.9

The incidence rate for osteomalacia per 1,000 mules during the period July 1, 1930, to July 1, 1931, was 73.9; the rate for horses was 268.3. The ratio of affected mules to horses was 1: 3.6, indicating that mules are far more resistant than horses to osteomalacia. Further evidence of this is shown by the fact that of 351 horses and 734 mules shipped to Fort Stotsenburg during the period 1927 to 1931 of which records are available, 15 per cent of the horses and only 4 per cent of the mules developed osteomalacia. The statement of Law(7) that mules habitually escape the disease did not apply in these animals.

Sex.—The Army purchases three geldings to one mare, approximately. Records showing the number of animals of each sex present in the command during the periods considered were unobtainable. Of the 366 cases recorded, 102 occurred among mares and 264 among geldings, which is a 1 : 2.6 ratio of affected mares to geldings. Since the geldings outnumber the mares by approximately three to one, it is believed that geldings and nonpregnant mares are affected equally. All investigators admit that pregnancy may influence the development and/or the course of the disease.

Age.—Analysis of the records indicates that osteomalacia may occur at any age beyond 4 years. Well-developed cases were noted in animals 25 years old. Table 5 shows the age of each case recorded, while fig. 5 shows the number of cases by age groups of two years and the percentage of total cases in each group.

Age ratios are not given because available records do not indicate the age of each animal in the command during the period considered.

Of the 366 cases recorded 266, or 73 per cent, occurred in animals of the age group 4 to 13 years. The age group 9 to 13 years included 210 cases, or 57 per cent of the total. The average age for all affected animals was 12.6 years; for affected horses, 12.1 years; and for affected mules, 13.4 years.

Comment.—1. The prevalence of osteomalacia is reported in Army animals in the Philippine Islands.

2. Diseases affecting the bones and organs of locomotion are more prevalent in Army animals in the Philippines than in like animals in the United States. The incidence of this group of diseases among the former showed a marked increase in 1924, approximately one year after a change of ration from imported to one composed largely of native feed.

3. It is believed that osteomalacia was prevalent in Army animals in the Philippines prior to its recognition in a large number of cases during 1930 and 1931. It was responsible for 23.7 per cent of all admission to the sick and wounded report at Fort Stotsenburg during the period July 1, 1930, to July 1, 1931.

4. It is believed that many conditions diagnosed as arthritis, ringbone, spavin, etc., were in reality osteomalacia.

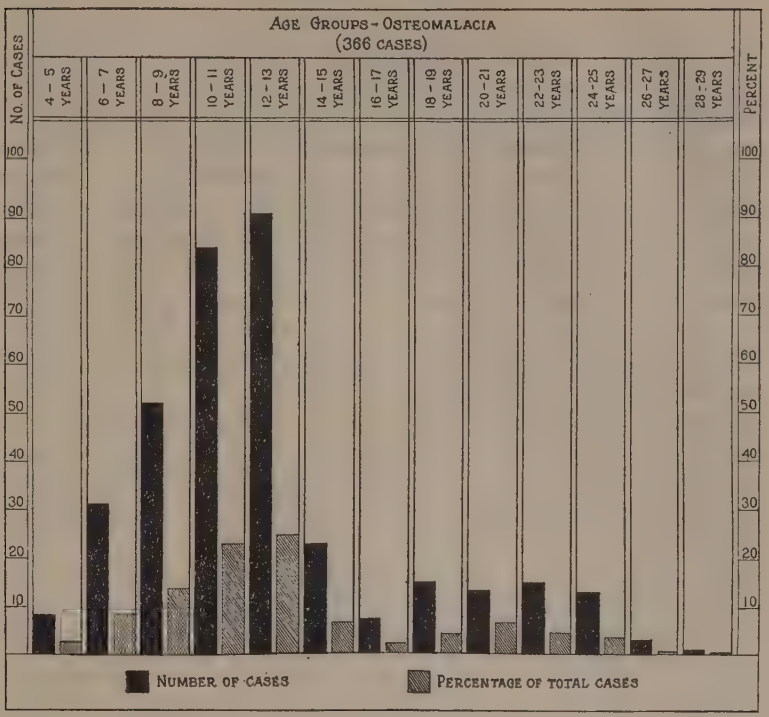

FIG 5. Number of cases and percentage of total in age groups of two years each.

5. Contrary to the opinion of some writers, osteomalacia affects mules as well as horses. The former animals, as compared with the latter, exhibit a marked resistance to the disease.

6. Osteomalacia may affect animals of all ages. At Fort Stotsenburg the condition was found to be prevalent particularly among animals from 6 to 13 years of age.

7. It is believed that sex is not a factor in the development and course of osteomalacia of equines.

ETIOLOGY

General.—The following conditions have been described as predisposing causes of osteomalacia in animals: Poor stable management, including lack of proper exercise and insanitary

conditions, insufficient sunlight, insufficient or improper feed, etc. Others believe that infections, toxæmias, heredity, etc., have an influence on the development of the disease. In the literature there is a wide diversity of opinion as to whether any of the conditions mentioned actually do predispose. Law(7) states that city environment is an important contributing factor in horses, and quotes Bern, who noted that cases removed from Brooklyn, New York, to the country usually recovered. Other investigators remark upon the high incidence of the disease among city horses as compared with animals maintained in the country. Statements of this character, however, are indefinite and details of animal management, feed, water, exercise, etc., usually are not given.

In the present investigation, dealing almost exclusively with Army animals, adverse conditions of management were not involved. These animals are maintained under excellent conditions. It was noted that hard work tended to increase the number of cases.

The multiplicity of exciting causes listed in the literature indicates the uncertainty that has existed as to the actual cause of the disease. Many investigators state that the cause is unknown. Among the many theories of cause advanced at various times are disturbances of internal secretion, parasitic infestation, heredity, infection, vitamin deficiency, and mineral deficiencies.

Disturbance of internal secretion.—Many writers believe in this theory, and it is claimed that different glands have been incriminated at one time or another. Winslow(6) states that Bossi has produced osteomalacia in sheep by removal of one suprarenal gland and that he has cured the condition in humans by injections of adrenalin. White(25) advances the theory that disturbances in ductless glands might reduce the alkalinity of the blood, thus permitting the lime salts to be dissolved. In various investigations of this disease, the ovary, parathyroid, and thymus have been held to be responsible.

In the present investigation, no gross abnormalities have been noted in the glands having internal secretions.

Parasitic infestation.—According to Roberts,(36) Conveur of Brazil believes that equine osteomalacia is due to cylicostome infestation. He found all cases to be grossly infested with this intestinal parasite and states that the administration of vermifuges resulted in improvement or recovery.

Heavy infestation with intestinal parasites was not noted in the present investigation; further, eosinophilia, a recognized indication of parasitism, was not demonstrated.

Heredity.—Bordeaux(9) in 1924 stated that, while the exact cause of osteomalacia of horses is unknown, inheritance and work are very strong predisposing factors. This writer suggests that heredity may have an influence and relates personal observations of six sires all of which produced a high percentage of offspring that developed the disease. Gonzales and Villegas(37) believe that osteoporosis of horses is a constitutional disease that is heritable.

That heredity may be a factor in the development of osteomalacia in some instances is conceivable when the work of Kramer and Howland,(38) Goldblatt,(39) and others is considered. The former investigators showed that a mineral defect in the diet is accentuated in succeeding generations of rats. Goldblatt demonstrated that rats whose parents had received cod-liver oil before and during pregnancy were better able to withstand the effects of deficient diets. The percentage of calcium in their bones was higher than in rats whose parents did not receive cod-liver oil. Both the above experiments indicate that mineral substances and vitamins may be stored in the offspring of animals.

In the present investigation there was no evidence that heredity exerted an influence in the development of the disease. Many typical cases appeared in animals over 20 years of age, and it is probable that the disease would have appeared long before this advanced age had been attained if heredity had exerted an influence.

Infection.—According to Hutyra and Marek,(12) this theory is finding more and more adherents. They state that Morpurgo, Moussu, Petrone, Pécaud, and others believe in this theory. Robertson(2) maintains that the condition is caused by a specific organism, although he obtained negative results on examination of blood and tissue specimens from affected animals. The contentions of this writer are based on an epizoölogical study of the disease in the Union of South Africa. Theiler(40) supports the theory of infection but failed to produce the disease by inoculation experiments. This investigator suggests that the microörganism or virus may be transmitted by insects. Lienaux(41) claims that osteomalacia may be produced by inoculation in small laboratory animals. Elliot(14) believes that the disease is due probably to an infection that enters the animal's

body through wounds. Law(7) states that Berns, Hoskins, and others have noted that "a fresh horse put in the stall of one that had suffered from osteoporosis soon contracted the disease." White(8) states that the disease is due probably to infection, as the change in the affected bones are of the character of infectious inflammations. Ingle(16) claims that the disorder is due to an infection that develops in animals rendered susceptible by defective diet. Henry(42) is opposed to the theory of infection, basing his opinion on the observation that affected cattle removed to nonaffected areas recovered from the disease. Mohler(1) quotes the experiments of Peason who obtained negative results by blood inoculations and bone transplants from affected to normal horses. Sturgess(10) and others do not support the theory of infection.

EXPERIMENTS

We observed that four of six advanced cases of osteomalacia under prolonged observation exhibited a moderate leucocytosis, intermittent in character and undetermined as to origin. This fact together with the epizoölogical study of the disease at Fort Stotsenburg, where a large number of cases were recognized in a short period of time, led the writers to investigate the theory of infection. This experiment was based on a bacteriological study of the disease, animal inoculation, and insect transmission.

Experiment 1.—March 1, 1931, the following animal was transferred to the board for experimental purposes: Species, horse; sex, gelding; age, 11 years; weight, 975 pounds; type, riding; color, roan. Diagnosis, osteomalacia, actively progressing type. Clinical examination confirmed the diagnosis. The animal showed a moderate shortening of the stride in all extremities. There were moderate proliferative changes along the lateral superior border of the rami of the mandible adjacent to the molar teeth, and slight enlargement of the facial bones. A "tucked-up" appearance of the flank was present and some deformity of the thorax was evidenced by a peculiar protruding appearance of the shoulder joints. The temperature was normal. The leucocyte count was 9,300, composed as follows: Monocytes, 2 per cent; lymphocytes, 47 per cent; neutrophylic granulocytes, 48 per cent (segmented, 40 per cent and "staff" or "band" forms, 8 per cent); eosinophiles, 2 per cent; mast cells, 1 per cent. The serum calcium was 12.5 milligrams per 100 cubic centimeters; and the inorganic phosphorus, 4.2 milligrams per 100 cubic centimeters. X-ray examination of the large metacarpal bone showed a slight rarefaction of the cortex. The animal was destroyed March 3 and autopsy confirmed the diagnosis.

Specimens of synovia from the humeroscapular and coxofemoral joints, bone marrow from the femur and humerus, and sections of bone from the metacarpals were collected under aseptic precautions. Blood specimens

were collected under aseptic conditions immediately prior to destruction of the animal. Aërobic and anaërobic cultures were prepared from these specimens using veal-infusion broth, veal-infusion agar, Nicolle-Novy-MacNeal media, dextrose broth, fresh beef-heart broth, cystine agar, and veal-infusion broth to which had been added small sections of osteomalacic bone. After forty-eight hours incubation at 37.5° F., the following aërobic and anaërobic cultures of the synovia, bone, and bone marrow showed the presence of a Gram-negative coccobacillus; dextrose broth, beef-heart broth, and veal-infusion broth containing bone. No growth developed in the blood specimen cultured. The organisms isolated from the synovia, bone marrow, and bone appeared to be identical.

Three cubic centimeters of a 48-hour veal-infusion-broth culture of this organism was injected intravenously into a normal horse. This animal was observed for a period of six months during which time no sign of osteomalacia appeared. Inoculation of the organism into rabbits, guinea pigs, white rats, and white mice gave negative results.

An agglutinating antigen was prepared from the organism by suspending it in physiological saline solution. Agglutination tests were made on the serum from five clinical cases of osteomalacia; negative results were obtained in all cases. A broth culture of the organism was used as an antigen for complement-fixation tests on five clinical cases of the disease; negative results were obtained in all cases. Ten days after inoculation of the horse, rabbits, and guinea pigs, with the coccobacillus, sera from these animals were tested for agglutinating and complement-fixing properties; negative results were obtained in all instances.

Experiment 2.—April 16, 1931, an advanced case of osteomalacia was destroyed and specimens of synovia and bone marrow collected for bacteriological examination. Aërobic and anaërobic cultures were prepared from these specimens, using blood-agar plates, veal-infusion broth, and beef broth to which small sections of osteomalacic bone had been added. Negative results were obtained with all specimens.

Experiment 3.—The intermittent leucocytosis exhibited by some cases of osteomalacia and for which no other cause was apparent, suggested the possibility of a "low-grade" infection. Repeated blood cultures were made from three cases of this type. In each instance specimens were collected on the day preceding a high leucocytic count. Aërobic and anaërobic cultures were prepared, using various culture media. Fig. 6 shows the type case under discussion and is representative of other cases that were cultured. Specimens were taken from this animal October 13, 17, 22, and 26, 1930. Negative results were obtained in all instances.

Experiment 4.—The suggestion by Theiler (40) that a virus or microorganism carried by insects might be responsible for osteomalacia, prompted the writers to undertake an experiment based on this theory. Two lots of laboratory-bred mosquitoes (78 female *Culex quinquefasciatus* and 92 female *Aëdes ægypti*) were allowed to feed on horse 1C09 October 13, 1930. This animal showed clinical symptoms of osteomalacia. On the day the insects fed the leucocytic count was 12,700. The leucocyte count on the following day was 13,450. After periods of seven and fourteen days these lots of mosquitoes were allowed to feed on a normal horse. The temper-

ature and blood count of this animal remained normal for a period of one month, and the animal was observed for a period of eight months. No signs of osteomalacia developed during this period.

Experiment 5.—October 23, 1930, 50 cubic centimeters of blood was removed from horse 1C09 and injected into the jugular vein of a normal horse. Animal 1C09 showed definite clinical symptoms of osteomalacia, and the leucocytic count was 12,700 on the day blood was collected for inoculation. The inoculated animal was observed for a period of five months; no sign of osteomalacia developed.

Comment.—As shown by the above experiments, no evidence was obtained indicating that equine osteomalacia as observed in the present investigation was caused by an infection.

Vitamin deficiency.—Following the discovery of vitamins, Funk(43) suggested that rickets was caused by lack of a specific vitamin responsible for normal mineral metabolism of bone. Mellanby(44) conducted the first experiments on the antirachitic food factor in 1919. Following the discovery that there is present in cod-liver oil a fat-soluble factor capable of influencing the deposition of calcium salts in the skeleton, considerable confusion existed as to the identity of this vitamin. Many investigators demonstrated that cod-liver oil contained a substance or substances capable of influencing growth and preventing xerophthalmia and influencing mineral metabolism. This substance was termed "vitamin A." In 1920, Hopkins(45) first demonstrated that, while vitamin A displayed a marked resistance to heat alone at a temperature up to 120° F., it was readily destroyed by oxidation. Later in the same year, Drummond and Coward(46) confirmed the work of Hopkins. This has since been confirmed by many investigators. In 1922, McCollum, Simmonds, and Becker(47) and McCollum, Simmonds, Becker, and Shipley(48) demonstrated conclusively that cod-liver oil contained a vitamin separate and distinct from the antixerophthalmia factor. These investigators showed that oxidized cod-liver oil did not cure xerophthalmia (vitamin A destroyed), but was equally effective in the cure of rickets as the unoxidized oil. This new substance was antirachitic or calcium depositing in its action and is now known as "vitamin D." Following the work of McCollum and others, many investigators demonstrated conclusively that vitamin D has a marked influence on calcium metabolism, and the lack of this factor is now believed to be the cause of human osteomalacia.

For many years investigators had noted that sunlight seemed to exert a beneficial effect in the prevention of diseases involv-

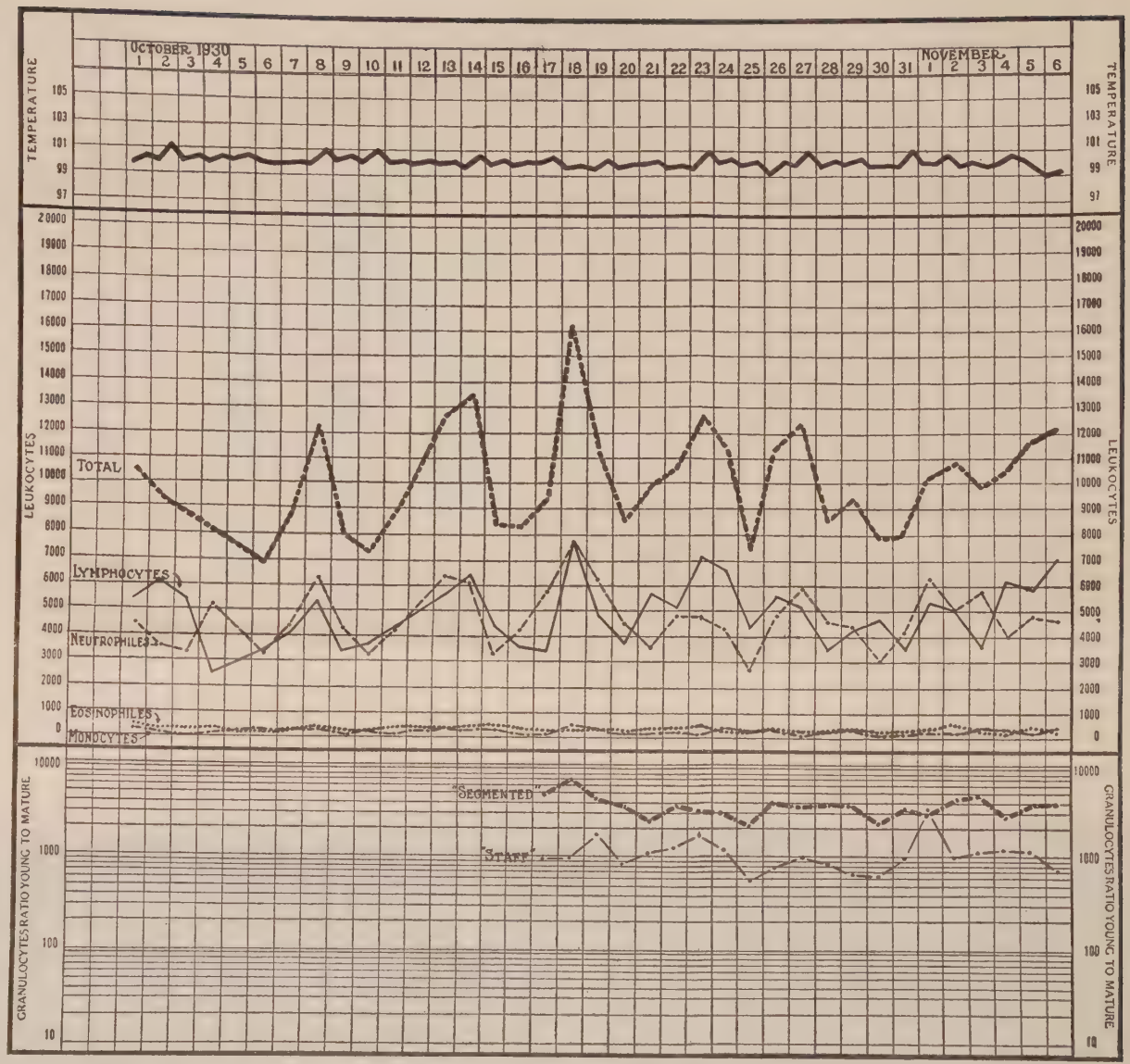

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FIG. 6. Leucocyte count and temperature; chart of horse 1C09.

ing the bones and in arresting these conditions. In 1924, Steenbock, Hart, and Jones(49) showed that direct sunlight improved the mineral metabolism of pigs. These investigators also demonstrated that while grains contained but little vitamin D, fresh green plant material is rich in this factor. In the same year Hess(26) showed that ultra-violet radiation of food imparted antirachitic properties to the substance so treated. Following the discovery that sunlight and ultra-violet rays stimulated the utilization of calcium in the animal body, many experiments have been conducted in an attempt to group or classify the species of animals that are benefited by these rays. In 1926, Hart, Steenbock, Elvehjem, and Scott,(50) working with milk cows, determined that sunlight had a feeble action on the utilization of calcium by these animals. In the same year, Eckels, Becker, and Palmer,(51) working with the same species of animal, suggested that the seasonal variation of mineral-deficiency diseases noted in these animals was probably due to variations in the ultra-violet content of sunlight. A year later, Hart, Steenbock, and Scott(52) found that ultra-violet rays had very little if any effect on the calcium and phosphorus metabolism of dairy cows. These writers suggest that in the cow the antirachitic vitamin is derived from the feed and in this respect the cow differs from man, goats, chickens, and probably rats.

Reid(17) believes that vitamin deficiencies are of little importance in diseases of animals. He states that investigators have shown that vitamins are frequently lost or destroyed during preparation of food for human consumption while this is not true of food consumed by animals.

While some investigators report beneficial results in equine osteomalacia by the administration of cod-liver oil, and most textbooks on the subject recommended its use, it has not proved to be beneficial in all cases. Sturgess(10) observed one case of the disease that was treated with one pint of cod-liver oil daily with no improvement. The concensus of opinion seems to be that vitamin D is an accessory factor in the etiology of equine osteomalacia.

EXPERIMENTAL

In order to determine the influence of vitamins on the course of equine osteomalacia and to test the efficacy of imported hay

and oats together with the influence of large amounts of fresh green grass, the writers conducted the following experiment:

Feeding experiment 1.—This was begun November 11, 1930, and the following animals, all of which exhibited symptoms of osteomalacia, were placed in a feeding group:

Horse B776.—Sex, gelding; color, bay; age, 13 years; weight, 710 pounds. Diagnosis of osteomalacia was made August 14, 1930. When examined November 11, the animal showed a marked shortening of the stride in all legs. There was a pronounced thickening of the rami of the mandible and slight proliferative changes of the facial bones. Some thoracic deformity was present as evidenced by a protruding appearance of the shoulder joints. The general condition was poor, and the hair coat of fair appearance. The serum calcium was 8.96 milligrams; and the inorganic phosphorus, 3.5 milligrams. X-ray examination of the metacarpal bone showed a moderate rarefaction and thinning of the cortex.

Horse B760.—Sex, gelding; color, brown; age, 12 years; weight, 750 pounds. Sick record showed a diagnosis of osteomalacia for this animal August 14, 1930. Clinically the animal showed a marked shortening of the stride in all four legs. There were marked proliferative changes of the mandible, and the facial bones showed a moderate enlargement. Slight deformity of the thorax was present (see Plate 1). The general condition and hair coat were fair. Serum calcium was 9.4 milligrams and inorganic phosphorus, 3.45 milligrams, per 100 cubic centimeters. The X-ray examination showed a slight rarefaction of the cortical portion of the metacarpal bone (see Plate 2).

Horse 249C.—Sex, gelding; color, black; age, 12 years; weight, 1,170 pounds. Sick record showed a diagnosis of osteomalacia for this animal November 10, 1930. The animal exhibited a moderate shortening of the stride in all legs. There were moderate proliferative changes of the bony tissue along the superior border of the rami of the mandible. No changes were noted in the facial bones. The general condition and hair coat were good. This animal showed a marked deformity of the thorax, exhibited by a protruding appearance of the shoulder joints and a sunken appearance of the sternum. The serum calcium was 9.45 milligrams and the inorganic phosphorus, 4.0 milligrams. X-ray examination of the metacarpal bone showed a moderate rarefaction of the compact portion.

Horse C442.—Sex, gelding; color, light chestnut; age, 11 years; weight, 870 pounds. Sick report showed a diagnosis of osteomalacia September 20, 1930. Clinical examination November 11, 1930, showed that this animal had a marked shortening of the stride in all four legs. There were marked proliferative changes of the rami of the mandible and enlargement of the facial bones. This animal showed the characteristic prominence of the shoulders and sunken appearance of the sternum (see Plate 3). The calcium content of the blood serum was 8.8 milligrams per 100 cubic centimeters; and the inorganic phosphorus, 3.96 milligrams. X-ray examination showed a moderate rarefaction of the metacarpal bone (see Plate 4, fig. 1).

Horse 74B9.—Sex, mare; color, dark chestnut; age, 7 years; weight, 950 pounds. Sick report showed a diagnosis of osteomalacia October 5,

1930. The animal showed a moderate shortening of the stride in the front legs while a slight shortening was present in the hind legs. There was a moderate thickening of the upper border of the rami of the mandible and moderate enlargement of the facial bones (see Plate 5). The serum calcium was 10.6 milligrams per 100 cubic centimeters; and the inorganic phosphorus, 5.64 milligrams. X-ray examination showed a slight rarefaction of the metacarpal bone (see Plate 6, fig. 1). The general condition and hair coat were good.

Animals B776, B760, and 249C were fed a basal ration composed of 2 pounds of American oats, 1 pound of crushed palay, 2 pounds of copra meal, 7 pounds of American grain hay, and 7 pounds of native rice hay. This ration is similar in composition to the ration fed regularly to Army animals in the Philippines with the exception that the green-grass component is omitted from the former ration. The nutritive ratio of the digestible crude protein and the combined digestible carbohydrates and fat (1 : 8.4) is within the limits prescribed for horses.(53) Chemical composition of products mentioned in all rations was computed from Table 19. During the feeding experiment, horse B776 received vitamins A and D in the form of cod-liver oil (United States Pharmacopœia), 300 cubic centimeters being administered daily. Horse B760 was given 10 cubic centimeters of Viosterol daily during the feeding period. No vitamins were added to the ration of horse 249C, this animal serving as control. The calcium-phosphorus content of this ration was CaO, 14.2, grams; P_2O_5, 33.4 grams; a ratio of 1 : 2.4.

While the primary object of this experiment was to determine the influence of vitamins A and D on the course of osteomalacia, it was thought desirable to determine the value of a large amount of green grass and the effect of a ration composed entirely of imported feed. Therefore, two animals were added to the feeding group:

Horse C442.—This animal received the same grain allowance as horses B776, B760, and 249C. The roughage portion of the ration was composed of American grain hay, 4 pounds; native rice hay, 4 pounds; and native green grass (commonly termed zacate), 18 pounds. The nutritive ratio of the ration was 1 : 7.7. The calcium-phosphorus content was CaO, 18.7 grams; P_2O_5, 45 grams; a ratio of 1 : 2.4.

Horse 74B9.—This animal was maintained on a ration composed of 5 pounds of American oats, 0.5 pound of American wheat bran, and 14 pounds of American grain hay. The nutritive ratio of this ration was 1 : 7.8. The calcium-phosphorus content was CaO, 13.6 grams; P_2O_5, 31.6 grams; a ratio of 1 : 2.3.

During the day all animals of the group were kept in a large corral where drinking water was available at all times. Chemical analysis of this water showed a calcium content of 19.5 parts per million (see Table 20). Salt in the form of compressed blocks was available to the animals at all times.

The progress of the disease is shown in detail in fig. 7.

While frequent clinical examinations of the experimental animals were of value, it was found that the course of osteomalacia could be followed with greater accuracy by X-ray examinations of the bones. (For details and technic, see page 72.) X-ray pictures of all experimental animals were made at frequent intervals for comparison, and this procedure furnished the most accurate means of determining whether the process was active, arrested, or healing.

As shown in fig. 7, slight clinical improvement was noted in the animal (B776) receiving cod-liver oil, and the pathological process in the bone, as shown by X-ray, was arrested apparently. The animal (B760) receiving vitamin D and the control animal (249C) both showed on clinical and X-ray examinations that the disease was active and progressing. An X-ray picture taken November 11, 1930, of the left metacarpal bone of horse B760 is shown in Plate 2, fig. 1; Plate 2, fig. 2, of the same animal was taken January 27, 1931. It will be noted in Plate 2, fig. 2, that the rarefaction was increased in the anterior part of the cortical portion and the posterior part of the compact bone shows a definite thinning.

The animal (C442) that received the green grass and the animal (74B9) maintained on imported feed, both showed definite evidence that the disease was progressing. Plate 4, figs. 1 and 2, shows the condition present in the metacarpal bone of horse C442, at the beginning and end of the experiment and Plate 6, figs. 1 and 2, shows the metacarpal bone of horse 74B9.

The serum calcium of the animal (B760) receiving Viosterol showed an increase of 17 per cent and that of animal (B776) receiving cod-liver oil showed an increase of 14 per cent; the control animal showed an increase of 8 per cent. The serum calcium of the animal (C442) receiving a large amount of green grass, showed an increase of 5 per cent and the animal (74B9) maintained on imported feed showed an increase of 4 per cent.

Comment.—In one case of equine osteomalacia, vitamins A and D supplied by 300 cubic centimeters of cod-liver oil daily for a period of seventy-seven days arrested the condition.

Animal No.	Ration	Added to Ration	Date	Physical Examination								Blood Chemistry (Milligrams per 100 cc Serum)		X-Ray Report
				Weight Lbs.	Head Lesions: Lower Mandible	Head Lesions: Facial	Shoulders	Lordosis	Gait: Front	Gait: Rear	General Condition	Coat	Calcium	Inorganic Phosphorus	
B776	Basic (1)	Vitamins "A and D" 300 cc Cod Liver Oil (U.S.P.) Daily	Nov. 11-1930	710	●●●	●	●		●●●	●●●	■■■	▲▲	8.96	3.51	●●
			Dec. 20-1930	690	●●●	●	●		●●●	●●●	■■	■■	10.20	4.00	●●
			Jan. 27-1931	710	●●●	●	●		●●	●●	■■	▲	10.20	4.17	●●
B760	do.	Vitamin "D" 10 cc Viosterol-250D Daily	Nov. 11-1930	750	●●●	●●	●		●●●	●●●	▲▲	▲▲	9.40	3.45	●
			Dec. 20-1930	750	●●●	●●	●		●●	●●●	■■	■■	10.90	4.00	●
			Jan. 27-1931	770	●●●	●●	●		●●●	●●●	■■	▲	11.00	3.33	●●
249C	do.		Nov. 11-1930	1170	●●		●●●		●●	●●	■■	■■	9.45	4.00	●●
			Dec. 20-1930	1170	●●		●●●		●●	●●●	▲▲	■■	10.03	4.00	●●
			Jan. 27-1931	1150	●●●		●●●		●●	●●●	▲▲	■■	10.20	3.23	●●●
C442	Special (2)		Nov. 11-1930	870	●●●	●●●	●●●	●	●●●	●●●	▲▲	▲▲	8.8	3.96	●●
			Dec. 20-1930	860	●●●	●●●	●●●	●	●●●	●●●	■■■	▲▲	8.0	4.00	●●
			Jan. 24-1931	850	●●●	●●●	●●●	●	●●●	●●●	■■■	▲▲	9.2	5.26	●●●
74B9	American (3)		Nov. 11-1930	950	●●	●●			●●	●	■■	■■	10.6	5.64	●
			Dec. 20-1930	950	●●	●●			●●		■■	■■	10.6	4.35	●●
			Jan. 24-1931	960	●●●	●●●			●●		■■	■■	11.0	4.56	●●

● = Slight or Mild
●● = Moderate
●●● = Marked or Severe

■ = Excellent
▲ = Very Good
■■ = Good
▲▲ = Fair
■■■ = Poor

(1) Basic Ration (daily)
- Oats, 2 lbs.
- Palay (crushed), 1 lb.
- Copra Meal, 2 lbs.
- American Grain Hay, 7 lbs.
- Rice Hay, 7 lbs.

(2) Special Ration (daily)
- Oats, 2 lbs.
- Palay (crushed), 1 lb.
- Copra Meal, 2 lbs.
- American Grain Hay, 4 lbs.
- Rice Hay, 4 lbs.
- Green Forage, 18 lbs.

(3) American Ration (daily)
- Oats, 5 lbs.
- Wheat Bran, ½ lb.
- American Grain Hay, 14 lbs.

W. O. 269252. Facing page 32.

Fig. 7. Feeding experiment 1.

Slight improvement was noted in the clinical symptoms, the serum calcium was increased 14 per cent, and no extension of the rarefaction was apparent in the metacarpal bone.

Vitamin D in the form of Viosterol, administered in 10 cubic centimeters doses daily for a period of seventy-seven days, failed to arrest the condition. Clinical symptoms showed no improvement, and the X-ray examination of the metacarpal bone showed an extension of the rarefaction. The serum calcium was increased 17 per cent.

Large amounts (18 pounds) of fresh green grass, fed for a period of seventy-four days, not only failed to arrest the condition but the disease progressed.

A ration composed of imported oats, wheat bran, and American grain hay failed to arrest the condition.

In the rations used, the ratio of calcium to phosphorus was between 1: 2.3 and 1: 2.4.

Mineral deficiency.—The theory of mineral deficiency as the cause of osteomalacia is supported by many investigators, but Mohler,(1) Theiler,(40) Robertson,(2) Elliot,(14) and others state that the condition is not caused by dietetic deficiencies. Of the many writers who support the theory of mineral deficiency, some believe that the disease is due to a lack of calcium in the diet or drinking water, others claim that a lack of phosphorus is responsible, while still others state that the condition is caused by an unbalanced ratio of the calcium and phosphorus intake. Henry(42) believes that a mineral deficiency is the cause and states that there is a correlation between soils deficient in lime and phosphoric acid and the prevalence of the disease. Ramsay(54) reports that soils from areas where osteomalacia is prevalent contain less nitrogen, lime, potash, and phosphoric acid than do soils from areas where the disease is uncommon. This writer states that grasses from affected areas contain less phosphoric acid and lime than do grasses from nonaffected areas and that the bones of animals reared in such surroundings contain less lime and phosphoric acid. Jensen(55) found a similar deficiency in soils from sections of New South Wales where the disease is prevalent. Tuff(20) quotes Müller who believes that the cause of osteomalacia is due to a deficiency of phosphoric acid in the fodder. Eckles, Becker, and Palmer(51) produced a mineral deficiency disease of cattle by feeding a ration deficient in phosphorus. Reid(17) states that osteoporosis of horses in India is observed frequently in animals feeding on soils that are

deficient in phosphorus. Friedberger and Frohner(5) believe that the cause of the condition is a deficiency of lime in the soil and fodder, and that water which is low in calcium content may assist development of the disease. Hutyra and Marek(12) agree. Van Saceghem(19) reports that, in districts of the Belgian Congo where the water contained little or no calcium, osteoporosis of horses occurred, whereas it never appeared in regions where the natural water contained sufficient calcium. This writer concludes that the cause of the disease is a deficiency of calcium. Niimi(56) and Niimi and Aoki(57) report that they were able to produce typical osteoporosis in horses by feeding a diet deficient in calcium. Control animals on the same diet (barley) to which was added calcium carbonate failed to develop the disease.

Zucker(58) states that Gerhard and Schlesinger studied (1899) the relation of the acid-base equilibrium in the animal body to the calcium and phosphorus metabolism. Ingle(16) in 1907 was the first to suggest the theory that osteoporosis of the Equidæ is favored by the use of foods, not necessarily deficient in lime and phosphates, but in which the ratio of lime and perhaps magnesium, to the phosphoric acid content, is too low. This writer states that the disease itself is caused probably by organisms that develop in animals rendered susceptible by a defective diet. Two years later the same writer(59) stated that, while osteoporosis of horses may be due to a specific organism, a condition of the bones similar to that which results from the disease may be induced by feeding a diet containing a low ratio of lime to phosphorus pentoxide. The statements of Ingle were based on chemical analyses of bones of affected animals as compared with normal bones. He maintains that the ratio of calcium to phosphorus in the feed should be about 1 : 1, basing this statement on the ratio of these elements in milk.

The numerous investigations of recent years that have been conducted with the view of determining the cause of human rickets have resulted in much evidence supporting the theory that a certain definite ratio between the calcium and the phosphorus intake must be maintained in order to prevent disturbances of mineral metabolism. The experiments of McCollum, Simmonds, Shipley, and Park,(60, 61) Sherman and Pappenheimer,(62) Kramer and Howland,(38) McClendon,(63) Orr, Holt, Wilkins, and Boone,(64) Mellanby and Killick,(65) Karelitz and Shohl,(66, 67) Shohl, Bennett, and Weed,(68) Hess,

Weinstock, and Rivkin,(69) Farquharson, Salter, and Aub,(70) and others have added much to the understanding of mineral metabolism and conditions that disturb this process. Practically all investigators now agree that the proportion of calcium to phosphorus in the diet is of prime importance in maintaining normal mineral metabolism of the skeleton. Constant increase in the amounts of either element will so disturb this ratio that harmful effects will result.

Forbes(3) showed that a deficiency of mineral bases in a ration will withdraw mineral matter from the bones. Tuff(20) maintains that for normal mineral metabolism not only sufficient quantities of minerals should be present in the forage but that there should be suitable quantitative proportions between the various minerals and between the acid and basic equivalents in the total fodder. He states that a large surplus of any one mineral or a difference greater than normal between the acid and basic equivalent in the fodder will dissolve minerals from the skeleton. Marek(4) agrees with this view and states that in herbivorous animals, the vegetable food must have a definite surplus of bases as compared with acids if no disturbance is to occur in the mineral metabolism. Of the more-recent work dealing with equine osteomalacia, that of Sturgess(10) deserves consideration. He produced osteomalacia in five horses by feeding diets low in calcium and high in phosphorus. The ratio of calcium oxide to phosphorus pentoxide ranged from 1:6.5 to 1:18. The control animal of this group received a ration in which the ratio of calcium to phosphorus was 1:2. This animal, after fourteen months, showed a "slight suspicion of commencing enlargement" of the inferior maxilla. The bone-meal component of the ration was then increased and the disease did not develop. He concluded that osteoporosis occurs when all possibility of infection by contact has been prevented and that, while a slight increase of phosphorus pentoxide (P_2O_5) over the calcium does not produce the disease, horses fed on diets low in calcium and high in phosphorus will develop osteomalacia.

During the present investigation cases of osteomalacia developed in animals which had been maintained on a ration composed of American oats, 3 pounds; crushed palay, 3 pounds; copra meal, 3 pounds; American grain hay, 4.5 pounds; native rice hay, 4.5 pounds; and green fodder, 12.5 pounds. The nutritive ratio of this ration was 1:7.6. Chemical analysis showed that the intake of calcium oxide was 18.2 grams and of phosphorus pentoxide 53.0 grams, a ratio of 1:2.9.

The following experiments were planned with a view to determining the influence of vitamin A on the course of osteomalacia and the importance of the ratio between the calcium and the phosphorus intake.

EXPERIMENTAL

In feeding experiment 1, it was found that equine osteomalacia could be arrested by supplementing the ration with vitamins A and D (cod-liver oil). The addition to the ration of vitamin D alone did not influence the progress of the disease. It seemed desirable, therefore, to determine the value of a ration containing products rich in vitamin A. In 1919 Steenbock(71) reported that yellow corn contains fat-soluble vitamin. The discovery that yellow corn is rich in vitamin A was confirmed later by Steenbock and Boutwell,(72) Steenbock and Coward,(73) Meyer and Hetler,(74) Russell,(75) and others. Therefore, yellow corn was added to the ration used in this experiment.

Chemical analysis of the ration on which Army animals in the Philippines were being maintained, showed that the calcium-phosphorus ratio was 1: 2.9. Osteomalacia developed in animals fed on this ration. In feeding experiment 1, the disease progressed in animals fed on a ration that contained calcium oxide and phosphorus pentoxide in the ratio of 1: 2.3 and 1: 2.4. In the following experiment sufficient finely ground limestone was added to the ration to give a calcium-phosphorus ratio of 1:0.9; the ration of the control animal had a ratio of 1:2.5.

Feeding experiment 2.—This was begun January 24, 1931, and continued for a period of three months. The following animals were placed in the feeding group:

Horse 3H70.—Sex, gelding; color, light chestnut; age, 9 years; weight, 860 pounds. Sick record showed a diagnosis of osteomalacia December 15, 1930. January 24, 1931, the animal showed a moderate ridge along the upper border of the mandible and slight enlargement of the facial bones. There was a shortening of the stride. The hair coat and general condition were fair. The serum calcium was 10.9 milligrams; and the inorganic phosphorus, 3.85 milligrams. X-ray examination of the metacarpal bone showed a moderate rarefaction of the compact portion.

Horse 76C3.—Sex, gelding; color, bay; age, 11 years; weight, 885 pounds. Clinical examination of this animal showed a slight thickening of the rami of the mandible. Slight shortening of the stride was present. The hair coat and general condition were fair. The serum calcium was 10.6 milligrams; and the inorganic phosphorus, 3.85 milligrams. X-ray examination showed a marked rarefaction of the cortex of the metacarpal bone. This case represents a type of osteomalacia that shows only slight symp-

toms on clinical examination but advanced changes in the bone (Plate 7, fig. 1).

Horse 38V0.—Sex, mare; color, bay; age, 4 years; weight, 760 pounds. The sick record showed a diagnosis of osteomalacia November 14, 1930. Moderate proliferative changes of the mandible and facial bones were present. Lordosis was present. The stride was shortened moderately. The general condition was fair, and the hair coat was good. The serum calcium was 11.1 milligrams; and the inorganic phosphorus, 4.76. X-ray examination showed a very slight rarefaction of the metacarpal bone.

Horse H463.—Sex, gelding; color, bay; age, 16 years; weight, 800 pounds. Sick record listed osteomalacia as a diagnosis January 6, 1931. Clinical examination January 24, 1931, elicited a moderate proliferation along the upper border of the mandible. Facial enlargement was absent. The stride was shortened. A moderate lordosis was present. The serum calcium was 11.3 milligrams; and the inorganic phosphorus, 3.57. X-ray examination showed a moderate rarefaction of the metacarpal bone.

Horse 1B11.—Sex, mare; color, dark bay; age, 12 years; weight, 820 pounds. Sick record gave a diagnosis of osteomalacia November 4, 1930. The animal was returned to duty November 22 and readmitted to sick report December 29, 1930, with a diagnosis of osteomalacia. January 24, 1931, the animal exhibited a moderate ridge along the superior border of the mandible. There was no facial enlargement. The stride was shortened moderately. The general condition was poor, and the hair coat fair. The serum calcium was 10.9 milligrams; and the inorganic phosphorus, 4.56. X-ray examination showed a slight rarefaction of the compact portion of the metacarpal bone.

Horse 13L2.—Sex, gelding; color, bay; age, 8 years; weight, 810 pounds. This animal was admitted to sick report with a diagnosis of osteomalacia (suspect) August 29, 1930, and returned to duty September 26, 1930. November 14, 1930, the animal was admitted to the veterinary hospital with a diagnosis of osteomalacia and returned to duty November 21, 1930. December 29, 1930, the animal was readmitted to the hospital with a diagnosis of osteomalacia. Clinical examination January 24, 1931, showed that the animal had a marked proliferation of bony tissue along the superior border of the rami of the mandible. There was a slight enlargement of the facial bones. The shoulders appeared to be displaced forward and there was a "tucked-up" appearance to the flank. The gait was shortened slightly. The hair coat was good, and the general condition fair. The serum calcium was 10.1 milligrams; and the inorganic phosphorus, 4.76. X-ray examination showed a slight rarefaction of the cortical portion of the metacarpal bone.

Horse C442.—This animal was used in feeding experiment 1, and the description of the animal together with the sick record has been given thereunder (Plates 3 and 4 and fig. 7). Clinical examination January 24, 1931, showed marked proliferative changes in the mandible and the facial bones. There was a marked prominence of the shoulder joints and a slight lordosis (Plate 3). The stride was shortened markedly, and there was a moderate "tucked-up" appearance to the flank. The general condition was poor and the hair coat fair in appearance. The serum calcium was 9.2 milligrams; and the inorganic phosphorus, 5.26. X-ray

examination showed a marked rarefaction of the compact portion of the metacarpal bone (Plate 4, fig. 2).

Horse 09V2.—Sex, gelding; color, bay; age, 20 years; weight, 750 pounds. Sick record of this animal showed an admission for osteomalacia June 20, 1930. The animal was returned to duty and readmitted for osteomalacia November 21, 1930. Clinical symptoms January 24, 1931, showed a marked change along the upper border of the rami of the mandible. No facial enlargement was present. A slight lordosis existed. The stride of the front legs was markedly shortened, and a moderate shortening was present in the hind legs. The general condition was fair, and the hair coat was of good appearance. The serum calcium was 11.8 milligrams; and the inorganic phosphorus, 3.9. X-ray examination showed a marked rarefaction of the compact portion of the metacarpal bone (Plate 8, fig. 1).

Horse 74B9.—This animal was used in feeding experiment 1, and the description of the animal and sick report have been given thereunder (fig. 7). Clinical examination January 24, 1931, showed marked proliferative changes in the mandible and the facial bones. The stride was shortened moderately in front. The general condition and hair coat were good. The serum calcium was 11.0 milligrams; and the inorganic phosphorus, 4.56. X-ray examination showed a moderate rarefaction of the metacarpal bone (Plate 6, fig. 2).

Horse 1B19.—Sex, gelding; color, bay; age, 12 years; weight, 740 pounds. The sick record of this animal showed an admission to hospital July 29, 1930, with a diagnosis of chronic gastroenteritis. September 1, 1930, malnutrition is recorded as a complication, and October 3, 1930, the entry of osteomalacia suspect was made. December 3, 1930, the diagnosis of osteomalacia was recorded. Clinically, January 24, 1931, the animal showed a slight enlargement of the mandible and slight lordosis. The stride was modified slightly. The flank showed the characteristic "tucked-up" appearance. The general condition and hair coat were good. The serum calcium was 10.7 milligrams; and the inorganic phosphorus 3.33. X-ray examination showed a slight rarefaction of the metacarpal bone.

Animals 3H70, 76C3, 38V0, H463, 1B11, 13L2, C442, 09V2, and 74B9 were fed a ration composed of oats, 2 pounds; yellow corn (cracked), 2 pounds; palay (crushed), 1½ pounds; copra meal, ⅔ pound; limestone (finely ground), 2.5 ounces; American grain hay, 4.5 pounds; native rice hay, 4.5 pounds; and fresh green grass, 12.5 pounds. The nutritive ratio of this ration was 1 : 9. The calcium oxide intake was 49 grams and the phosphorus pentoxide, 42 grams, a ratio of 1 : 0.9.

Horse 1B19 was added to the feeding group as a control and received a ration composed of oats, 2 pounds; palay (crushed), 2 pounds; copra meal, 2 pounds; American grain hay, 4.5 pounds; native rice hay, 4.5 pounds; and fresh green grass, 12.5 pounds. The nutritive ratio of this ration was 1 : 8. The intake of calcium oxide was 17 grams and of phosphorus pentoxide, 43 grams, a ratio of 1 : 2.5.

During the day all animals of the group were kept in a large corral where drinking water was available at all times. Chemical analysis of this water showed a calcium content of 19.5 parts per million.

Fig. 8 shows the progress of the disease in all animals of this group. It will be noted, by reference to fig. 8, that all animals of the group showed improvement except the control animal (1B19). Five of the nine animals on the test ration were returned to light duty, while one animal (74B9) was returned to full duty. The disease showed definite progression in the control animal (1B19). Plate 7, figs. 1 and 2, shows the change that took place in the metacarpal bone of horse 76C3. Plate 8, fig. 1 shows the metacarpal bone of horse 09V2 January 24, 1931, and Plate 8, fig. 2, April 24, 1931.

Animal Number	Date	Physical Examination						Blood Chemistry (Milligrams Per 100 cc Serum)		X Ray Examination	Remarks
		Weight (Lbs.)	Head Lesions: Mandible	Head Lesions: Facial	Gait: Front	Gait: Rear	General Condition	Calcium	Inorganic Phosphorus		
3H70	1931 Jan. 24	860	●●	●	●●	●●●	▲▲	10.9	3.85	●●	
	April 24	880	●●	●	●●	●●	■	11.8	4.00	●	
76C3	Jan. 24	885	●			●	▲▲	10.6	3.85	●●●	
	April 24	910	●			●	■	12.2	3.45	●●	Light duty
38V0	Jan. 24	760	●●	●●		●●	▲▲	11.1	4.76	●	
	April 24	810	●●	●●●			■	13.0	3.87	Normal	Light duty
H463	Jan. 24	800	●●		●	●	▲▲	11.3	3.57	●●	
	April 24	870	●●			●	■	10.8	3.87	●	Light duty
1B11	Jan. 24	820	●●		●●	●●	■■■	10.9	4.56	●	
	April 24	880	●●		●		■	12.9	3.87	● (improved)	Light duty
13L2	Jan. 24	810	●●●	●	●	●	▲▲	10.1	4.76	●	
	April 24	880	●●	●			■	14.6	3.13	Normal	Light duty
C442	Jan. 24	850	●●●	●●●	●●●	●●●	■■■	9.2	5.26	●●●	
	April 24	850	●●●	●●	●●	●●	▲▲	9.8	4.00	●	
09V2	Jan. 24	750	●●●		●●●	●●	▲▲	11.8	3.94	●●●	
	April 24	770	●●		●		▲	11.0	4.00	●	
74B9	Jan. 24	960	●●●	●●●	●●		■■	11.0	4.56	●●	
	April 24	930	●●●	●●●			■	12.7	3.87	●●	Full duty
1B19 (Control)	Jan. 24	740	●			●	■■	10.7	3.33	●	
	April 24	790	●●		●	●	▲	12.5	3.13	●●	

● .Slight or mild
●● .Moderate
●●● .Marked or severe
■ .Excellent
▲ .Very good
■■ .Good
▲▲ .Fair
■■■ .Poor

Test Ration		Control Ration	
Oats	2 lbs.	Oats	2 lbs.
Yellow corn	2 lbs.	Palay	2 lbs.
Palay	1⅓ lbs.	Copra meal	2 lbs.
Copra meal	⅔ lbs.	American grain hay	4½ lbs.
Limestone (finely ground)	2½ oz.	Rice hay	4½ lbs.
American grain hay	4½ lbs.	Green grass	12½ lbs.
Rice hay	4½ lbs.		
Green grass	12½ lbs.		

Fig. 8. Feeding experiment 2.

At the beginning of the experiment, the average serum calcium for all animals fed on the test ration was 10.8 milligrams and the inorganic phosphorus 4.35 milligrams per 100 cubic centimeters. These values at the end of the experiment were 12.1 and 3.78 milligrams, respectively, an increase of 12 per cent in serum calcium and a decrease of 10.8 per cent in inorganic phosphorus. It will be noted in the section on chemical pathology that a low serum calcium and a high serum inorganic phosphorus are present in osteomalacia.

Comment.—1. Nine horses, affected with osteomalacia and maintained for ninety days on a ration to which yellow corn and limestone had been added, showed definite improvement clinically, and X-ray examination of the metacarpal bones showed that the disease had been arrested and an increased deposition of minerals had taken place. The mineral ratio of the ration was calcium oxide 1 to phosphorus pentoxide 0.9.

2. Changes in the serum calcium and inorganic phosphorus indicated that improvement of the condition had taken place.

3. One control animal maintained on a ration having a ratio of calcium oxide to phosphorus pentoxide of 1 : 2.5 showed no clinical improvement. X-ray examination showed progressive rarefaction of the bone.

Feeding experiment 3.—This experiment was started April 24, 1931, and continued for a period of sixty days. The animals composing the group were the same as in feeding experiment 2, and the ration the same with the exception that the limestone was omitted in the present experiment. The nutritive ratio remained unchanged, but the intake of calcium oxide was 14.2 grams (instead of 49 grams) and of phosphorus pentoxide, 42 grams, a ratio of approximately 1 : 3 (instead of 1 : 0.9). Control animal 1B19 received a ration having a ratio of calcium oxide to phosphorus pentoxide of 1 : 2.5.

As shown in fig. 9, the disease progressed in eight of the nine animals. The average serum calcium for the group remained approximately the same. The inorganic phosphorus increased about 6 per cent.

Comment.—A ration composed of oats, yellow corn, crushed palay, copra meal, American grain hay, native rice hay, and green fodder failed to arrest equine osteomalacia. On the other hand the condition progressed in eight of the nine animals on test. The calcium-phosphorus ratio was 1 : 3, approximately.

The condition progressed in the control animal, which was fed a ration containing calcium and phosphorus in the ratio of 1 : 2.5.

Animal Number	Date	Physical Examination						Blood Chemistry (Milligrams per 100 cc Serum)		X Ray Examination
		Weight (lbs)	Head Lesions: Mandible	Head Lesions: Facial	Gait: Front	Gait: Rear	General Condition	Calcium	Inorganic Phosphorus	
3H70	1931 April 24	880	●●	●	●●	●●	■	11.8	4.00	●
	June 24	890	●●	●	●●	●●●	▲	12.2	3.85	●●
76C3	April 24	910	●			●	■	12.2	3.45	●●
	June 24	900	●●		●●		▲	12.8	3.85	●●●
38V0	April 24	810	●●	●●●			■	13.0	3.87	Normal
	June 24	820	●●	●●●	●		▲	12.2	3.85	●
H463	April 24	870	●●			●	■	10.8	3.87	●
	June 24	860	●●		●	●	▲	11.2	3.85	● (Progressed)
1B11	April 24	880	●●		●		■	12.9	3.87	●
	June 24	880	●●		●		■	12.2	4.00	● (Progressed)
13L2	April 24	880	●●	●			■	14.6	3.13	Normal
	June 24	820	●●	●			■	12.6	3.57	Normal
C442	April 24	850	●●●	●●	●●	●●	▲▲	9.8	4.00	●
	June 24	820	●●●	●●●	●●●	●●●	■■■	10.4	5.00	●●
09V2	April 24	770	●●		●		▲	11.0	4.00	●
	June 24	760	●●		●		■■	12.2	4.35	●●
74B9	April 24	930	●●●	●●●			■	12.7	3.87	●●
	June 24	890	●●●	●●●			■	12.2	3.57	●●●
1B19 (control)	April 24	790	●●		●	●	▲	12.5	3.13	●●
	June 24	780	●●		●	●	▲	12.2	4.35	●● (Progressed)

● = Slight or mild
●● = Moderate
●●● = Marked or service
■ = Excellent
▲ = Very good
■■ = Good
▲▲ = Fair
■■■ = Poor

Test Ration		Control Ration	
Oats	2 lbs	Oats	2 lbs.
Yellow corn	2 lbs.	Palay	2 lbs.
Palay	1⅓ lbs.	Copra meal	2 lbs.
Copra meal	⅔ lbs.	American grain hay	4½ lbs.
American grain hay	4½ lbs.	Rice hay	4½ lbs.
Rice hay	4½ lbs.	Green grass	12½ lbs.
Green grass	12½ lbs.		

Fig. 9. Feeding experiment 3.

Feeding experiment 4.—This experiment was started July 22, 1931, and continued for a period of ninety-eight days. The animals were placed in three groups of five each as follows:

Group A.

Horse 76C3.—This animal had been used in feeding experiments 2 and 3. Clinically, it showed a moderate thickening of the rami of the man-

dible. There was a moderate shortening of the stride. The serum calcium was 11.3 milligrams; and the inorganic phosphorus, 4.35 milligrams. The general condition was very good. X-ray examination of the metacarpal bone showed a marked rarefaction of the cortex.

Horse O9V2.—This animal had been used in the two previous feeding experiments. Clinically, it showed moderate proliferative changes of the mandible. The stride was shortened slightly, and the general condition was good. The serum calcium was 12.0 milligrams; and the inorganic phosphorus, 4.76 milligrams. X-ray examination of the metacarpal bone showed a rarefaction of moderate degree.

Horse H463.—This animal had been used in feeding experiments 2 and 3. Examination showed a slight thickening of the rami of the mandible. There was a slight shortening of the stride. The general condition was very good. The serum calcium was 11.5 milligrams; and the inorganic phosphorus, 4.7 milligrams. The metacarpal bone showed a slight rarefaction on X-ray examination.

Horse 1B11.—This animal had been used in feeding experiments 2 and 3. Clinically, there was a moderate proliferative change of the mandible. The stride was shortened slightly, and the general condition was excellent. The serum calcium was 11.6 milligrams; and the inorganic phosphorus, 4.0 milligrams. X-ray examination of the metacarpal showed a slight rarefaction of the cortex.

Horse OC35.—Sex, gelding; color, brown; age, 10 years; weight, 820 pounds. Sick record showed that this animal had been admitted to the veterinary hospital at Fort Stotsenburg December 13, 1930, with a diagnosis of ringbone, right foreleg. The animal remained in the hospital until December 23, 1930, when it was returned to duty. January 30, 1931, a diagnosis of osteomalacia was made. When examined July 22, 1931, the animal showed a moderate enlargement of the mandible and a slightly shortened stride. The general condition was good. The serum calcium was 11.3 milligrams; and the inorganic phosphorus, 4.7 milligrams. X-ray examination showed a slight rarefaction of the metacarpal bone.

Group B.

Horse B776.—This animal had been used in feeding experiment 1. Clinically, there was manifested a marked thickening of the rami of the mandible and slight proliferative changes of the maxillæ. The stride was shortened markedly, and the general condition was fair. The serum calcium was 10.6 milligrams; and the inorganic phosphorus, 4.76 milligrams. X-ray examination of the metacarpal bone showed a marked rarefaction (Plate 9, fig. 1).

Horse 249C.—This animal had been used previously in feeding experiment 1. Clinically, it showed a moderate enlargement of the rami of the mandible and a moderately shortened stride. The general condition was fair. The serum calcium was 10.5 milligrams; and the inorganic phosphorus, 4.0 milligrams. X-ray examination showed a marked rarefaction of the metacarpal bone.

Horse C442.—This animal had been used previously in feeding experiments 1, 2, and 3. There was a marked enlargement of the rami of the mandible and marked proliferative changes of the facial bones. The stride was shortened markedly, and the general condition was poor. The

serum calcium was 10.0 milligrams; and the inorganic phosphorus, 4.35 milligrams. X-ray examination showed a marked rarefaction of the metacarpal bone.

Horse 1B19.—This animal had been used in feeding experiments 2 and 3. July 22, 1931, it showed a moderate enlargement of the rami of the mandible and a slightly shortened stride. The general condition was very good. The serum calcium was 12.5 milligrams; and the inorganic phosphorus, 5.0 milligrams. X-ray examination of the metacarpal bone showed a moderate rarefaction of the cortical portion.

Mule B059.—Sex, gelding; color, gray; age, 11 years; weight 950 pounds. The sick record of this animal showed a diagnosis of osteomalacia May 7, 1391. Clinically, there was a moderate proliferative change along the rami of the mandible and a shortening of the stride. The general condition was very good. The serum calcium was 11.0 milligrams; and the inorganic phosphorus, 4.17 milligrams. X-ray examination showed a slight rarefaction of the metacarpal bone.

Group C.

Mule H223.—Sex, mare; color, brown; age, 7 years; weight, 970 pounds. The sick record showed a diagnosis of osteomalacia March 13, 1931. The animal had continued on a duty status until admitted to the hospital for this condition April 9, 1931. The record shows hospital treatment from April 9 to June 24, 1931, when the animal was transferred to Manila for experimental purposes. There was present a slight ridge along the upper border of the rami of the mandible and slight proliferative changes of the facial bones. The stride was shortened slightly, and the general condition was very good. The serum calcium was 12.0 milligrams; and the inorganic phosphorus, 4.76. X-ray examination of the metacarpal bone showed a slight rarefaction of the cortical portion.

Horse 3H70.—This animal had been used previously in feeding experiments 2 and 3. Moderate proliferative changes along the upper border of the mandible and slight enlargement of the facial bones were present. The stride was shortened, and the general condition was very good. The serum calcium was 12.4 milligrams; and the inorganic phosphorus, 5.0. X-ray examination showed moderate rarefaction of the metacarpal bone.

Horse L698.—Sex, mare; color, chestnut; age, 12 years; weight, 780 pounds. The sick record showed an admission to the veterinary hospital at Fort Stotsenburg January 29, 1931, with a diagnosis of osteomalacia. The animal was returned to duty February 28, 1931, readmitted for osteomalacia June 8, 1931, and transferred to Manila June 24, 1931. Clinical examination showed marked proliferative changes along the superior border of the rami of the mandible and a moderate shortening of the stride. The general condition was good. The serum calcium was 11.8 milligrams; and the inorganic phosphorus, 4.0. X-ray examination showed a slight rarefaction of the cortex of the metacarpal bone.

Horse 278L.—Sex, mare; color, brown; age, 8 years; weight, 800 pounds. The sick record showed a diagnosis of osteomalacia January 28, 1931, the animal being marked off duty January 31, 1931. March 21, 1931, the animal was admitted to the hospital with the same diagnosis and continued in that status until transferred to Manila for experimental purposes June 24, 1931. Clinically, the animal showed moderate proli-

ferative changes along the superior border of the rami of the mandible and slight enlargement of the facial bones. The stride was shortened markedly, and the general condition was good. The serum calcium was 10.8 milligrams; and the inorganic phosphorus, 4.35. X-ray examination showed a slight rarefaction of the metacarpal bone.

Horse 5B42.—Sex, mare; color, chestnut; age, 11 years; weight, 840 pounds. A diagnosis of osteomalacia was recorded in May, 1931. Examination July 22, 1931, showed slight proliferative changes along the upper border of the rami of the mandible. The stride was shortened moderately, and the general condition was fair. The serum calcium was 11.8 milligrams; and the inorganic phosphorus, 4.17. X-ray examination showed a moderate rarefaction of the cortical portion of the metacarpal bone.

RATION

Group A.

The animals of this group (76C3, O9V2, H463, 1B11, and OC35) were fed a ration composed of oats, 3 pounds; crushed palay, 3 pounds; copra meal, 2 pounds; American grain hay, 4.5 pounds; native rice hay, 4.5 pounds; and green grass, 12.5 pounds. The nutritive ratio was 1 : 8.7. A sufficient amount of commercial calcium chloride was added to the drinking water to increase the calcium content to approximately 400 parts per million. During an observation period of ten days it was found that these animals would drink approximately 6.5 gallons of water daily. The total mineral intake was calcium oxide 32 grams and phosphorus pentoxide 48 grams, a ratio of calcium to phosphorus of 1 : 1.5. August 6 it was noted that the animals of this group were not consuming their entire allowance of 8 pounds of concentrates. This allowance was then reduced to 6 pounds, resulting in a nutritive ratio of 1 : 8.3 and a mineral intake of calcium oxide of 31.6 grams and phosphorus pentoxide 43 grams, a ratio of calcium to phosphorus of 1 : 1.4. All animals of the group were exercised from one to two hours daily during the test period.

Group B.

The animals of this group (B776, 249C, C442, 1B19, and B059) were fed a ration composed of oats, 1.5 pounds; crushed palay, 1.5 pounds; copra meal, 1 pound; American grain hay, 4.5 pounds; native rice hay, 4.5 pounds; and green grass, 12.5 pounds. Each animal received 36 grams of finely ground limestone daily, the analysis of which showed 46.56 per cent calcium calculated as the oxide. The calcium content of the drinking water was 19.5 parts per million. The nutritive ratio of this ration was 1 : 8.7. The total mineral intake was calcium oxide 34 grams and phosphorus pentoxide 36 grams, a ratio of approximately 1 : 1. The animals of this group were kept in a large corral during the day and stabled at night. No exercise was given.

Group C.

The animals of this group (H223, 3H70, L698, 278L, and 5B42) were fed the same ration as were the animals of group B with the exception that each animal received 0.5 pound of wheat bran daily, and 100 grams calcium phosphate was substituted for the finely ground limestone (calcium carbonate). The nutritive ratio remained approximately the same (1 : 8.5). The calcium oxide intake was the same as in group B (34

grams) and the phosphorus pentoxide 85 grams, a ratio of 1 : 2.5. Animals of this group were kept in a large corral during the day and stabled at night; no forced exercise was given.

The progress of the disease in the above groups of animals is shown in fig. 10. It will be noted, by reference to fig. 10, that all animals of group A showed improvement. The X-ray examination showed that the condition had been arrested, and there was evidence that repair of the bone was taking place. The average serum calcium at the beginning of the experiment was 11.5 milligrams; and the inorganic phosphorus, 4.5 milligrams. At the completion of the experiment the animals of this group showed an increase of 11 per cent in the serum calcium and a decrease of 18 per cent in the inorganic phosphorus. It has been found, as shown under the section on chemical pathology, that in active equine osteomalacia the serum calcium is reduced and the inorganic phosphorus is increased. The intake ratio of calcium oxide to phosphorus pentoxide was 1 : 1.4.

The calcium intake of animals of groups B and C was approximately the same (34 grams). The addition of ground limestone to group B animals resulted in a calcium-phosphorus ratio of 1 : 1, and the addition of calcium phosphate to the ration of group C animals gave a ratio of 1 : 2.5. It will be noted, by reference to fig. 10, that all animals of group B showed definite improvement while three of the five animals of group C showed that the disease was progressing. The serum calcium of the animals of group B was increased 16 per cent, and the inorganic phosphorus was decreased 31 per cent. The averages for serum calcium and inorganic phosphorus of the animals of group C was approximately the same at the completion of the experiment as at the beginning.

Comment.—Osteomalacia was arrested and the condition showed evidence of healing in all cases(5) fed on a ration in which the calcium-phosphorus ratio was 1 : 1.4.

Two groups of five cases each were fed rations containing the same amounts of calcium but different quantities of phosphorus. All animals of the group receiving a ration in which the calcium-phosphorus ratio was 1 : 1 showed definite improvement. The other group was fed a ration in which the calcium-phosphorus ratio was 1 : 2.5, which was produced as a result of the use of calcium phosphate instead of calcium carbonate. In three of the five animals of this group, definite progression of the disease was noted.

Animal Number	Date	Physical Examination: Weight (Lbs.)	Head Lesions: Mandible	Head Lesions: Facial	Gait: Front	Gait: Rear	General Condition	Blood Chemistry (Milligrams per 100 cc serum): Calcium	Blood Chemistry (Milligrams per 100 cc serum): Inorganic Phosphorus	X Ray Examination
Ration "A"										
76C3	1931 July 22	890	●●		●●		▲	11.3	4.35	●●●
	Oct. 28	880	●●		●	●	▲	11.5	3.57	●●● (Improved)
09V2	July 22	750	●●		●	●	■■	12.0	4.76	●●
	Oct. 28	730	●		●	●	■■	12.9	3.85	●
H463	July 22	850	●		●	●	▲	11.5	4.70	●
	Oct. 28	860	●		●	●	▲	13.1	4.00	● (Improved)
1B11	July 22	900	●●		●		■	11.6	4.00	●
	Oct. 28	900	●		●		■	13.1	3.57	● (Slightly Improved)
0C35	July 22	820	●●		●		■■	11.3	4.70	●
	Oct. 28	810	●				■	13.4	3.70	● (Improved)
Ration "B"										
B776	July 22	720	●●●	●	●●●	●●●	▲▲	10.6	4.76	●●●
	Oct. 28	740	●●	●	●●●	●●	▲▲	13.4	3.33	●●
249C	July 22	1090	●●		●●	●●	▲▲	10.5	4.00	●●●
	Oct. 28	1110	●●		●	●	■■	13.1	2.86	●●● (Improved)
C442	July 22	810	●●●	●●●	●●	●●●	■■■	10.0	4.35	●●●
	Oct. 28	820	●●●	●●	●●	●●	▲▲	11.7	3.03	●●
1B19	July 22	770	●●		●	●	▲	12.5	5.00	●●
	Oct. 28	790	●				▲	14.0	3.49	●● (Improved)
B059	July 22	950	●●		●	●●●	▲	11.0	4.17	●
	Oct. 28	980	●●		●	●●	■	12.5	2.78	● (Improved)
Ration "C"										
H223	July 22	970	●	●	●	●	▲	12.0	4.76	●
	Oct. 28	980	●	●		●	▲	12.3	4.00	●
3H70	July 22	910	●●	●	●	●●	▲	12.4	5.00	●●
	Oct. 28	920	●●	●	●●	●●	▲	12.8	4.76	●● (Progressed)
L698	July 22	780	●●●		●●	●●	■■	11.8	4.00	●
	Oct. 28	770	●●●		●●	●●	▲▲	12.1	4.70	●
278L	July 22	800	●●	●	●●●	●●●	■■	10.8	4.35	●
	Oct. 28	790	●●	●	●●	●●●	■■	12.0	4.00	●●
5B42	July 22	840	●		●●	●●	▲▲	11.8	4.17	●
	Oct. 28	810	●●		●●●	●●●	■■	10.2	5.00	●●

● . Slight or mild ■ . Excellent ▲▲ . Fair
●● . Moderate ▲ . Very good ■■■ . Poor
●●● . Marked or severe ■■ . Good

Fig. 10. Feeding experiment 4.

Feeding experiment 5.—Out of a shipment of animals arriving in the Philippines from the United States in July, 1930, thirty-one horses were shipped to Fort Stotsenburg where they were fed the regular ration for Army animals. This ration was oats, 3 pounds; crushed palay, 3 pounds; copra meal, 3 pounds; American grain hay, 4.5 pounds; native rice hay, 4.5 pounds; and native green grass, 12.5 pounds. The nutritive ratio was 1 : 7.3. The drinking water supplied these animals contained 9.7 parts of calcium per million. The total intake of calcium oxide per animal was 18.5 grams, and the phosphorus pentoxide, 53 grams, a ratio of 1: 2.9.

Twenty-five horses from the above-mentioned shipment were kept at Nichols Field and fed a ration of American oats, 9 pounds, and American grain hay, 14 pounds. The nutritive ratio was 1 : 7.6. The drinking water at this station contained 33 parts of calcium per million. The total calcium oxide intake was 15.7 grams, and the phosphorus pentoxide intake was 29.1 grams, a ratio of 1 : 1.9. The total mineral intake in all cases was based on the mineral content of the ration plus the minerals contained in 6.5 gallons of water, the latter being the average amount consumed by each animal daily. All animals were of the riding type and their ages ranged from 4 to 7 years. Several days after their arrival in Manila, complete physical examinations were made and the serum calcium and inorganic phosphorus determined. No evidence of osteomalacia was found. Both groups were examined at frequent intervals during the next nine months. The serum calcium and inorganic phosphorus determinations were made after a period of approximately six months.

Veterinary hospital records at Fort Stotsenburg show that three, approximately 10 per cent, of the thirty-one animals shipped to that station developed osteomalacia within nine months. The first case occurred in March, 1931, in an 8-year-old bay mare. This animal was destroyed in June, 1931, after hospitalization for ninety-one days. The second case occurred in a 5-year-old bay mare and was diagnosed April 16, 1931. This animal was not hospitalized and continued on a duty status. The third animal of this group that developed the condition was a 6-year-old bay mare. This case was diagnosed osteomalacia May 11, 1931, and was returned to duty after eighteen days in the hospital.

Frequent clinical examinations of the twenty-five animals kept at Nichols Field failed to demonstrate the presence of osteomalacia in this group.

As previously stated, serum calcium and inorganic phosphorus determinations were made on all animals of each group at the time of their arrival in the Philippines, and again approximately six months later. Table 9 shows the serum calcium and inorganic phosphorus values in animals of each group at the time of arrival in the Philippines and six months later.

It will be noted that very little change had taken place in the serum calcium and the inorganic phosphorus of the animals fed a ration in which the calcium oxide-phosphorus pentoxide ratio was 1 : 1.9, whereas the group of animals maintained on a ration in which this ratio was 1 : 2.9 showed a decrease

of approximately 5 per cent in the serum calcium and an increase of approximately 20 per cent in the inorganic phosphorus. The writers have found that the serum calcium is decreased and the inorganic phosphorus is increased in groups of animals showing osteomalacia. Fig. 11 shows graphically the changes noted.

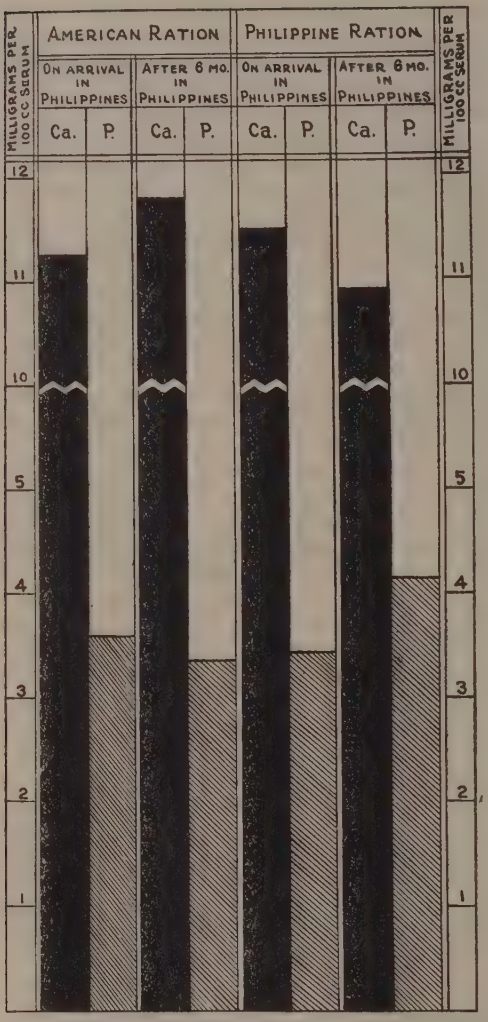

Fig. 11. Comparison of American and Philippine rations.

Comment.—In this feeding experiment, approximately 10 per cent of a group of thirty-one animals fed on a ration in which the calcium-phosphorus intake ratio was 1:2.9 developed clinical osteomalacia within nine months.

A reduced serum calcium and an increased inorganic phosphorus were present in this group.

No evidence of osteomalacia was found in a group of twenty-five horses whose calcium-phosphorus intake ratio was 1:1.9.

No blood changes indicative of osteomalacia were evident in this group.

EPIZOÖLOGICAL STUDY

In conjunction with the etiology of osteomalacia, certain pertinent facts were evident in an epizoölogical study, details of which are given elsewhere in this article. Prior to 1923, all Army animals in the Philippines were fed a

TABLE 9.—*Calcium and phosphorus content of serum of American horses in the Philippine Islands.*

American ration.					Philippine ration.				
Animal No.	Milligrams per 100 cc serum.				Animal No.	Milligrams per 100 cc serum.			
	On arrival in P. I.		After 6 mo. in P. I.			On arrival in P. I.		After 6 mo. in P. I.	
	Calcium.	Inorganic phosphorus.	Calcium.	Inorganic phosphorus.		Calcium.	Inorganic phosphorus.	Calcium.	Inorganic phosphorus.
5	10.5	3.23	11.5	3.33	1	10.3	2.94	10.8	4.00
8	11.1	3.33	13.2	3.23	2	11.6	3.45	11.6	4.56
9	12.8	3.70	11.1	3.33	3	12.1	3.33	11.0	4.76
10	12.3	3.85	11.2	4.00	4	11.5	2.94	10.8	5.00
17	10.5	3.70	12.2	3.57	6	12.3	3.57	11.4	4.17
19	11.1	3.70	13.4	3.13	11	13.2	3.33	11.5	4.00
21	10.2	3.85	12.2	3.57	13	13.5	3.23	10.2	4.35
28	11.7	4.35	12.2	3.45	14	13.7	3.85	11.2	3.85
29	12.1	4.00	11.7	3.85	15	14.0	3.13	10.6	4.00
32	10.6	3.70	11.5	2.86	20	11.3	3.03	12.3	4.00
33	11.7	3.33	11.2	3.13	23	11.3	3.33	10.8	4.76
34	12.6	3.85	11.5	3.13	26	10.9	4.00	10.6	4.35
35	11.2	5.00	11.3	3.70	27	11.8	3.70	10.6	3.85
37	11.6	3.57	11.9	3.33	31	10.2	2.63	10.2	3.70
39	10.5	3.85	11.9	3.13	36	11.0	3.33	11.2	4.76
41	11.9	3.13	11.6	3.85	42	10.6	3.57	10.2	4.35
44	10.5	3.13	11.4	3.23	43	10.8	3.85	10.8	3.70
48	10.2	3.13	11.2	3.45	45	11.2	3.13	10.4	4.00
54	11.0	3.85	11.7	3.45	47	12.9	3.13	9.8	4.00
55	10.6	3.45	11.2	3.13	49	10.2	3.85	11.2	4.00
59	11.0	3.13	12.6	3.23	50	10.2	4.56	11.2	4.17
60	11.2	3.03	11.2	3.33	51	10.9	4.00	10.2	4.00
65	13.0	3.85	11.7	3.33	52	10.6	3.85	10.8	3.33
67	11.4	3.13	11.3	3.13	53	10.2	3.45	10.6	4.00
69	10.9	3.33	12.4	3.57	57	10.8	3.13	12.6	3.33
					58	10.9	3.85	11.7	3.70
					61	11.0	3.03	11.2	3.85
					62	11.2	3.13	11.0	4.55
					63	11.1	3.13	10.6	3.45
					64	11.1	3.13	10.8	4.55
					68	12.7	3.33	10.8	4.76
Average	11.3	3.61	11.8	3.34	Average	11.5	3.42	10.9	4.12

ration composed of oats and American grain hay. Available records for this period fail to show the presence of osteomalacia, and the incidence rate for diseases of the bones and organs of locomotion approximates the rate for Army animals in the United States. While available data do not show the calcium

and phosphorus intake at that time, recent analyses indicate a calcium-phosphorus ratio of approximately 1: 1.9.

In 1923 when the ration was changed to oats, crushed palay, copra meal, American grain hay, native rice hay, and green grass, the calcium-phosphorus ratio that resulted was approximately 1 : 2.9. Following this change of ration the incidence rate for diseases of the bones and of the organs of locomotion showed a sharp increase.

From 1923 to July, 1931, all Army animals in the Philippines received approximately the same ration. Study of the available records of animal conditions at the larger Army posts for the period July 1, 1930, to July 1, 1931, showed that there was a striking difference between the osteomalacia rates at several of the posts. The fact that the ration at all posts was identical led the writers to investigate the composition of the drinking water supplied. Chemical analyses showed a wide variation in the calcium content of the water at the different posts (see Table 20). It was found that osteomalacia did not occur at Fort Mills (Corregidor) where approximately one hundred animals were kept. The drinking water at this post showed the highest calcium content of any post in the Philippines; namely, 125 parts per million. Based on an average daily consumption of 6.5 gallons of water per animal and the ration fed, the calcium oxide-phosphorus pentoxide intake ratio was 1 : 2.3. At Fort Stotsenburg the water contained the lowest calcium content (9.7 parts per million) and the records show the highest incidence of osteomalacia. Based on the same amount of water consumed daily and the same ration, the calcium oxide-phosphorus pentoxide intake ratio of animals at Fort Stotsenburg was 1 : 2.9.

Further evidence that the cause of osteomalacia in the present investigation was due to a wide calcium-phosphorus ratio is shown by the striking reduction in the number of cases that occurred at Fort Stotsenburg following a change of ration in July, 1931, when finely ground limestone was added to the ration and the calcium-phosphorus intake ratio was reduced to approximately 1 : 1.

CONCLUSIONS

Based on the above described feeding tests and the evidence presented in an epizoölogical study of osteomalacia during the

present investigation, it is believed that the following conclusions may be drawn:

No evidence was obtained which indicated that equine osteomalacia is due to disturbances of internal secretions, parasitic infestations, heredity, or infections. Evidence of vitamin deficiencies was not noted.

The production of equine osteomalacia was due to a ration containing a large amount of phosphorus in proportion to the calcium intake of the animal. It has been shown that the amount of calcium intake is of secondary importance to the calcium-phosphorus ratio. Osteomalacia will develop in animals when the ratio of calcium oxide to phosphorus pentoxide is 1 : 2.9. The condition did not develop during a nine-month period when this ratio was 1 : 1.9. Evidence is presented showing that the calcium content of the drinking water was of particular importance.

Feeding experiments with affected animals showed that the condition was arrested and anabolic processes stimulated when the ratio of the calcium oxide-phosphorus pentoxide intake was 1 : 0.9, 1 : 1.0, and 1 : 1.4. The condition progressed in affected animals when this ratio was 1 : 2.3, 1 : 2.4, 1 : 2.5, and 1 : 3.

PATHOLOGY

Macropathology.—In 1905, Friedberger and Fröhner(5) gave a classical description of the macroscopic picture of the bones of animals suffering from osteomalacia, saying that the condition manifested itself chiefly as a decalcification and softening of the bones from within outward accompanied by hyperemia and with the conversion of the bony tissue into a fibrous, soft mass, there being a fatty degeneration and atrophy of the bone cells together with an enlargement of the medulla and a thinning of the cortex. They noted that the change varies with the severity of the affection and that, on close examination of slight cases, there is an increased blood supply and enlarged blood vessels may be seen in the bones. Their sections showed "small dots of blood," the marrow being permeated by numerous small extravasations. They also called attention to the fact that the inner surface of the cortex showed numerous small bony plates detached from the cortical mass. They found that in severe cases the medulla increased to such an extent that it penetrated into the epiphyses and that the marrow became soft, gelatinous, even watery, and had a "dirty yellow color." Law(7) in 1911

reported that on the articular ends of affected bone, granulation tissue penetrates into and through the articular cartilages and that the changes are greatest in the bones of the face. Many writers note that inflammatory reactions are present frequently in the joints of animals affected with osteomalacia. Goldberg(76) in 1917 pointed out that inflammation of the joints of horses are commoner than is generally supposed, appearing in the joints of apparently healthy animals in numerous instances and that the lesions most frequently found in these cases are fibrous outgrowths on the synovial membrane and erosions on the articular surfaces. The writers have noted these lesions in apparently normal animals of advanced age. From these observations it would appear that too much importance cannot be attached to the finding of these lesions in osteomalacia.

Careful study of osteomalacia as exhibited in horses and mules in the Philippines has convinced the writers that all bones of the skeleton are involved in the process, and that the variation in the degree of severity in certain parts of the skeleton is due, probably, to external influences such as mechanical irritation, etc. An illustration of this is the enlargement of the mandible which is due probably to irritation consequent to mastication. Initial gross changes in nearly all instances were noted in bones which have a thin cortex normally such as the mandible. A certain amount of deformity of this bone was noted in practically all cases, there being a thinning and softening of the cortex together with a honeycomb appearance of the medullary portion. In about 5 per cent of the cases similar deformities were found in the facial bones. In many of the cases observed there was a more or less severe involvement of the vertebræ, there being an extreme thinning of the cortex and intervertebral discs. The long bones showed an increase of the medulla at the expense of the cortex. Many detached or partially detached portions of the cortex were noted at the periphery of the medulla. The cortical portion of long bones showed a porosity varying with the severity of the condition. Porosity was found to be much more severe in thick cortices. The outer portion of the cortex showed punctiform hæmorrhages in nearly all instances. These frequently involved the periosteum as well.

In all cases autopsied, severe joint lesions were noted. There were erosions on the articular surfaces of the bones entering into the formation of joints, a thickening of the synovial membrane and, in some cases, the presence of an organized dark yellow exudate.

The marrow of the long bones examined was found to be softer than normal, fatty in appearance, and varied in color from dark yellow to chocolate. The severity varied in different portions of the same bone. Petechial hæmorrhages were common. The autopsy protocol of horse B760 is a typical case.

HOSPITAL RECORD OF HORSE B760

Horse B760.—The following hospital record was obtained: February 21, 1929, admitted to veterinary hospital, Fort Stotsenburg. Diagnosis, tendinitis, acute, flexor tendons, right front. Returned to duty February 23, 1929. November 14, 1929, admitted to veterinary hospital. Diagnosis, contused wound, sole of right front foot. The record shows that January 31, 1930, the animal developed an intertubercular bursitis of the right shoulder. June 27, 1930, the animal was returned to duty. August 14, 1930, admitted to the veterinary hospital with a diagnosis of osteomalacia. September 25, 1930, the animal was transferred to Manila and from November 11, 1930, to January 27, 1931, was used in feeding experiment 1. Clinical examination May 11, 1931, showed a marked enlargement of the mandible and moderate proliferative changes of the facial bones. A slight lordosis was present, and the shoulder joints were prominent. There was a marked shortening of the stride (Plates 1 and 2). The animal was destroyed May 11, 1931.

PROTOCOL

General condition.—The cadaver was thin but not emaciated. Ribs prominent. External examination showed no apparent softening of the bones. Skin, subcutaneous tissue, and musculature were normal apparently.

External examination.—1. Head and neck: A marked bilateral thickening of the mandibular body, together with a rounding of the superior border adjacent to the molar teeth. The nasal bones were enlarged moderately, and the nasal passages were reduced.

2. Trunk: Slight lordosis, prominent shoulder joints, and sunken appearance of the breast.

3. Extremities: Apparently normal.

Internal examination.—1. Head and neck: Thyroids and parathyroids appeared normal. The cortical portion of the rami of the mandible showed thinning in certain areas due, apparently, to an invasion by cancellous and osteoid tissue; islands of soft bone were seen in the remaining cortex. A characteristic ridge formation appeared on the outer surface of both rami of the mandible. There was a thinning of the cortex of the maxillæ. All bones heretofore described were cut easily with the autopsy knife.

2. Thorax: Pericardium and pericardial fluid normal, there being a normal quantity of the latter. Lungs and pleura showed no gross pathology except hypostatic congestion of the lower lobe of the right lung. The heart was normal in size and weight, the musculature and valves being normal. Sections of the vertebral column showed an abnormal relationship between the medulla and cortical bone in that only a shell of the latter remained. The intervertebral discs were thinned. The spinous and transverse processes were softened to such an extent that they were cut readily with a knife (see Plate 13). The spinal cord and fluid appeared normal.

3. Abdomen: Pancreas, adrenals, stomach, kidneys, and liver appeared to be normal. There was no gross pathology evident in the intestines or peritoneum. Serous fluid was normal in quantity and color.

Extremities.—Articular surfaces of the hip joints were involved extensively by an erosive process and erosions of lesser severity were found in all other joints of the extremities (see Plate 12). The erosive processes appeared to be subacute but sufficient to account for the lameness manifested clinically. The joint capsules were thickened markedly. There was an invasion of the joint capsule by a tissue, embryonal in appearance. There was no enlargement of any of the bones of the extremities, but the cortical portion was thinned and showed punctiform hæmorrhages in many places. The marrow was dark brown to chocolate in areas where the process was most severe. On fracture, the long bones showed a marked porosity of the cortex.

The histopathology of this case is given in detail in the following section.

Micropathology.—Wells(27) claims that absorption depends upon a removal of both organic and inorganic substances by osteoclasts followed by the formation of uncalcified osteoid tissue. Hutyra and Marek(12) state that the spaces between the bony pillars as well as the Haversian canals are often seen to be distended and bulging. Most of the bony pillars are ossified only in the middle, the sides of their walls next the marrow having patchlike or, more often, lamellar-formed osteoid borders, sometimes so decalcified that lacunar bulgings with polynuclear giant cells (osteoclasts) are present. Law(7) states that there is an active proliferation of cells in the Haversian systems and marrow spaces and an increased vascularity.

For the study of the micropathology of osteomalacia as exhibited in our cases, specimens were collected from the following case and forwarded to the Army Medical Museum, Washington, D. C.(77) for examination.

Horse B760, heretofore mentioned as animal 2 under Feeding Experiment 1, age 12 years. Diagnosis of osteomalacia August 14, 1930. Autopsy May 11, 1931. Clinical manifestations of the disease were typical at the time of autopsy. Protocol of the autopsy on this animal will be found under macropathology (see Plates 1 and 2). A section of the mandible lateral to the molar teeth showed a tumor mass, cutting easily with a knife but containing lime salts. Under low magnification, the section showed a layer of fibrous tissue overlying a fibrous cortex. Under higher magnification, the cortex of the bone was composed of fibrous tissue. Beneath this, and at right angles to it, were partially decalcified lamellæ. Between them was dense fibrous tissue. Beneath this was a zone where most of

the lamellæ had been replaced by fibrous tissue, this zone being approximately 2 millimeters in width. Below this, there was somewhat less destruction of the bony lamellæ throughout the rest of the section. There appeared to be much more fibrous tissue than the interlamellar spaces would normally hold, and this suggested that the bony enlargement was due to the fibrous tissue overgrowth. In areas of bone destruction, giant cells were numerous.

Gross and microscopic appearance of the maxillary bone was the same as that of the mandible, but in the former the process seemed slightly less advanced.

The heart muscle was normal apparently.

A section from the head of the femur showed erosions of the articular surface. The section, as cut, was roughly triangular, with the articular cartilage covering the hypotenuse of the triangle. At the base and apex of this triangular portion the bone approached the normal, as seen under low power, with some fatty marrow between the lamellæ of bone. In the midportion of the triangle there was a loss of substance of most of the lamellæ, with a disappearance of some, the changes becoming less toward the base and apex.

Under higher magnification, low power, there was evident a disorganization of the bone, and a replacement with fibrous connective tissue. The earliest change was evidenced by an erosion of the bony trabeculæ, numbers of foreign body giant cells appearing along the eroded bone. At the same time, there appeared to be a loss of calcium salts in some of the lamellæ. The marrow was gradually replaced by new cellular fibrous tissue of the osteoid type beginning along the edges of the lamellæ, and gradually occupying the marrow space. It also appeared that the continued advance of the process replaced the entire lamellar structure with this fibrous tissue. The articular cartilage over the area where the greatest bone destruction had occurred showed fewer nuclei, and stained peculiarly, the surface appearing somewhat eroded. This was a definite degeneration, but here normally the cartilage is of somewhat different consistency, due to the attachment of joint ligaments.

The section from the bone and marrow of the middle third of the large metacarpal showed that, near the marrow cavity, fibrous tissue was replacing partially decalcified bone, and infiltrating marrow spaces nearest the marrow cavity. This was evidently an early change, and consistent with the diagnosis of osteomalacia, though doubtless there were no gross evidences

of the widening of the marrow cavity. This probably would have occurred had the animal lived long enough.

Bone marrow from the proximal end of the femur was fatty in character and showed active hæmopoiesis, which may or may not have been in excess of the normal.

Photomicrographs pertinent to the above-mentioned descriptions are reproduced.

Chemical pathology.—General.—Certain dietetic requirements are necessary for the animal body to maintain normal mineral metabolism and deficiencies or even variations in these requirements may produce disturbances of metabolism and result in clinical manifestations of disease. These requirements include, among others, a sufficient supply of vitamin D, calcium, and phosphorus. The present study of equine osteomalacia has confirmed the opinion of other investigators that the relative proportion of the calcium and phosphorus intake is of prime importance in maintaining normal mineral metabolism of the skeleton. Evidence of disturbed mineral metabolism is indicated, to a certain degree, by the amount of mineral substances the animal retains and by the chemical composition of the blood and of the bones.

Many investigators have demonstrated that variation in the proportion of calcium to phosphorus in the ration will result in the retention of different amounts of these elements. Meigs and Turner(78) showed that the phosphorus assimilation of cows may be reduced by an excess of two or more parts of calcium in the ration to one part of phosphorus. The experiments of Forbes(79) showed that the addition of calcium carbonate or of pulverized limestone to the ration of swine caused a marked increase in the retention of calcium, magnesium, and phosphorus. Orr, Holt, Wilkins, and Boone,(64) experimenting with children, demonstrated by metabolism studies that (*a*) excessive amounts of calcium in the diet increased the total calcium absorption and retention but impaired the phosphorus retention and (*b*) excessive amounts of phosphorus in the diet exercised an unfavorable influence on the calcium metabolism and a larger amount of calcium is lost in the fæces. They believe that the retention of one element in the intestines by an excessive amount of the other in the diet is due to the formation of insoluble phosphates. Karelitz and Shohl(67) showed that a change from a high calcium-low phosphorus intake to a ratio of 1 : 1 resulted in an increased calcium retention. Marek(4) states that the degree of retention of calcium depends upon its ratio with other

mineral constituents as well as upon its mutual quantitative ratio to proteins, fats, and carbohydrates. Shohl, Bennett, and Weed(68) state that the ratio of calcium to phosphorus in retention is proportional to the ratio of the same elements in the diet.

Blood chemistry.—As osteomalacia is a disease marked by disturbed metabolic activity, it seemed desirable to ascertain if this activity was reflected by changes in the normal content of the blood constituents. A review of available literature failed to show the results of complete blood chemistry on normal horses. Hayden and Tubangui(80) reported the following results in blood chemical determinations on seven normal horses: Nonprotein nitrogen, 34 milligrams; urea, 17.8 milligrams; uric acid, 2.45 milligrams; creatinin, 1.80 milligrams; and sugar, 106 milligrams per 100 cubic centimeters of blood. The most complete work recorded is that of Holt and Reynolds(81) who, in 1924, determined the hæmoglobin, sugar, urea nitrogen, nonprotein nitrogen, uric acid, and creatinin on a group of normal horses. Their results are shown in Table 10.

TABLE 10.—*Blood chemistry in osteomalacia.*

Constituent.	Cases.	Low.	High.	Average.
Hæmoglobin (Dare or Tallquist method) per cent	136	70.0	98	88.2
Sugar do	141	0.078	0.37	0.109
Urea nitrogen mg	141	7.50	25.0	14.81
Nonprotein nitrogen mg	141	16.50	42.58	25.82
Uric acid mg	138	1.76	6.66	2.76
Creatinin mg	141	0.9	3.0	1.4

Hayden and Fish(82) published in the Report of the New York State Veterinary College, Cornell University (1927–28), the following results on normal horses: Sugar, 102.9 milligrams; urea, 18.7 milligrams; uric acid, 2.45 milligrams; nonprotein nitrogen, 33.9 milligrams; creatinin, 1.81 milligrams.

In the present investigation, normal values for blood constituents were determined on sixty-nine horses that arrived in the Philippines from the United States July 10, 1930. To avoid any possible influence that Philippine forage might have on the determinations, this group was fed American oats and American grain hay during the test period. Complete physical examination by two veterinary officers showed that all animals of the group were normal apparently. The following technic was employed in collecting the specimens:

Blood from the jugular vein was collected before the morning feed. In order to facilitate the determinations, groups of ten

animals were bled daily. Specimens for the nonprotein nitrogen, urea nitrogen, creatinin, uric acid, sugar, chlorides, and iron were collected in 50 cubic centimeter tubes containing 20 milligrams of dry sodium oxalate. For the calcium, inorganic phosphorus, and magnesium determinations, specimens of blood were collected in large centrifuge tubes, allowed to clot, and the serum separated by centrifugalization. After collection of the specimens they were taken to the laboratory and the determinations made with the least possible delay. The determinations for nonprotein nitrogen, urea nitrogen, creatinin, uric acid, sugar, and chlorides were made on the protein-free blood filtrate prepared according to the method of Folin and Wu.(83)

The methods of Folin and Wu(83) were used in the nonprotein nitrogen and creatinin determinations. The method for sugar was the improved Folin and Wu(84) method. For the determination of urea nitrogen, the Folin and Wu method, as modified by Craig,(85) was used. The method of Benedict(86) was used for the determination of uric acid. Chlorides were determined by the Whitehorn(87) method. The determination of iron was made according to the method of Wong.(88) The hæmoglobin was calculated from the iron determination. The method for serum calcium determination was that of Kramer and Tisdall as modified by Tisdall(89) and further modified by Clark and Collip.(90) The inorganic phosphorus was determined by the Briggs(91) modification of the Bell Doisy method. The magnesium content was determined on the supernatant liquid from the calcium determination after centrifugalization, plus the first wash fluid, according to the method of Briggs.(92)

Table 11 shows the determination on the above-mentioned group of normal horses and gives the average, maximum, and minimum values for each constituent.

Similar determinations were then made on thirty-six horses showing typical symptoms of osteomalacia. The results of these determinations are given in Table 12.

Analysis of Table 12 shows that the hæmoglobin, nonprotein nitrogen, urea nitrogen, creatinin, uric acid, sugar, chlorides, iron, and magnesium of osteomalacia cases are within the normal limits established, approximately. The serum calcium in the osteomalacia cases shows a reduction of approximately 9 per cent. The inorganic phosphorus of the serum shows an increase of 20 per cent.

The ration on which animals developed osteomalacia was exceedingly high in its phosphorus content as compared with its

TABLE 11.—*Blood chemical determinations on normal horses.*

Animal No.	Percentage of hæmoglobin (calculated).	Milligrams per 100 cc blood.							Milligrams per 100 cc serum.		
		Non-protein nitrogen.	Urea nitrogen.	Creatinin.	Uric acid.	Sugar.	Chlorides as NaCl.	Iron.	Calcium.	Inorganic phosphorus.	Magnesium.
1	74	22.2	13.0	2.00	2.50	80	446	42	10.3	2.94	1.70
2	65	21.4	13.5	1.00	2.00	77	462	37	11.6	3.45	2.24
3	65	23.6	12.5	2.00	3.00	80	462	37	12.1	3.33	2.12
4	67	22.2	13.0	1.00	3.00	74	404	38	11.5	2.94	1.74
5	90	21.4	13.2	1.00	3.00	67	380	51	10.5	3.23	1.70
6	62	21.4	12.5	1.00	4.00	83	371	35	12.3	3.57	2.06
7	74	27.2	13.0	1.00	3.00	94	429	42	11.3	2.86	2.01
8	74	21.0	14.2	1.50	3.00	94	429	42	11.1	3.33	1.96
9	63	21.2	13.7	2.00	2.00	80	462	36	12.8	3.70	2.06
10	69	31.6	13.0	2.28	2.84	74	429	39	12.3	3.85	1.96
11	63	26.1	13.7	2.28	2.10	91	446	36	13.2	3.33	1.78
12	62	23.1	15.8	1.56	2.00	80	446	35	12.9	4.17	2.24
13	65	31.6	12.5	1.65	2.84	83	462	37	13.5	3.23	2.12
14	94	33.3	13.7	2.00	3.63	67	413	53	13.7	3.85	1.91
15	65	30.0	12.5	2.28	2.67	100	479	37	14.0	3.13	1.91
16	69	31.6	18.8	1.56	2.84	91	462	39	14.3	4.00	1.57
17	94	24.0	13.6	2.00	2.66	100	429	53	10.5	3.70	2.45
18	90	27.3	13.0	1.76	2.58	87	446	51	10.9	4.17	2.24
19	99	25.0	13.6	1.81	3.07	100	462	56	11.1	3.70	2.00
20	99	27.3	16.7	1.87	2.22	83	446	56	11.3	3.03	1.85
21	86	28.6	12.5	1.87	2.85	80	462	49	10.2	3.85	2.18
22	79	33.3	11.1	2.14	2.66	87	479	45	11.1	4.00	2.30
23	99	27.3	10.3	2.00	2.00	100	462	56	11.3	3.33	2.18
24	90	28.6	11.1	2.00	2.85	80	429	51	10.7	4.00	1.91
25	83	31.6	12.0	2.00	2.00	83	462	47	11.1	4.17	1.70
26	79	30.8	11.5	1.79	2.05	83	462	45	10.9	4.00	1.57
27	79	33.3	12.0	1.57	2.66	100	479	45	11.8	3.70	1.78
28	76	34.0	13.0	1.57	2.75	95	446	43	11.7	4.35	1.63
29	85	27.3	11.5	1.79	2.96	83	446	48	12.1	4.00	1.57
30	95	32.0	12.0	1.79	2.27	81	413	54	11.5	4.17	1.88
31	74	26.0	13.0	2.00	2.50	91	479	42	10.2	2.63	1.63
32	85	29.3	15.0	1.87	2.42	105	462	48	10.6	3.70	1.63
33	71	27.3	16.7	1.57	3.81	100	413	40	11.7	3.33	1.40
34	88	33.3	21.4	1.76	2.05	95	363	50	12.6	3.85	1.57
35	76	28.6	14.3	1.76	2.00	95	462	43	11.2	5.00	1.60
36	78	33.3	18.3	1.66	2.11	100	495	44	11.0	3.33	1.48
37	104	28.6	13.0	1.50	2.22	91	446	59	11.6	3.57	1.57
38	99	28.6	13.0	1.57	2.22	91	446	56	10.7	3.33	1.88
39	85	30.0	14.3	1.57	2.16	118	446	48	10.5	3.85	1.63
40	79	31.6	13.6	1.87	2.50	118	462	45	11.8	3.85	1.78
41	78	32.5	13.3	1.90	3.20	95	462	44	11.9	3.13	1.74
42	69	30.8	14.3	1.80	4.00	87	495	39	10.6	3.57	1.70
43	85	30.0	12.8	1.76	3.33	83	479	48	10.8	3.85	1.70
44	67	36.0	14.3	1.76	2.33	111	495	38	10.5	3.13	1.48
45	67	35.3	12.5	1.76	3.08	118	462	38	11.2	3.13	1.42
46	74	24.0	12.5	1.66	3.20	100	479	42	10.8	3.13	1.51
47	67	28.6	17.6	1.76	3.08	91	446	38	12.9	3.13	1.67
48	56	30.0	16.7	1.66	2.33	100	479	32	10.2	3.13	1.57
49	86	28.6	15.4	2.72	3.08	87	429	49	10.2	3.85	1.51

TABLE 11.—*Blood chemical determinations on normal horses*—Continued.

Animal No.	Percentage of hæmoglobin (calculated).	Milligrams per 100 cc blood.							Milligrams per 100 cc serum.		
		Non-protein nitrogen.	Urea nitrogen.	Creatinin.	Uric acid.	Sugar.	Chlorides as NaCl.	Iron.	Calcium.	Inorganic phosphorus.	Magnesium.
50	79	32.5	15.0	1.76	3.08	83	462	45	10.2	4.56	1.35
51	85	27.3	17.2	1.57	2.10	71	446	48	10.9	4.00	1.40
52	76	29.0	17.0	1.42	2.00	80	462	43	10.6	3.85	1.33
53	99	26.8	16.8	1.66	2.00	83	495	56	10.2	3.45	1.40
54	99	33.3	15.4	1.57	3.30	95	446	56	11.0	3.85	1.40
55	76	37.5	18.0	1.76	2.10	91	446	43	10.6	3.45	1.35
56	86	28.6	17.0	1.76	3.48	83	429	49	10.8	4.17	1.51
57	76	28.6	14.3	2.30	4.00	133	495	43	10.8	3.13	1.51
58	74	26.0	13.6	2.30	3.81	125	462	42	10.9	3.85	1.40
59	88	30.0	16.7	2.14	3.20	133	446	50	11.0	3.13	1.40
60	85	33.3	15.0	2.14	3.08	100	429	48	11.2	3.03	1.40
61	74	28.6	13.6	2.00	3.33	125	495	42	11.0	3.03	1.40
62	78	31.6	15.8	2.00	4.00	95	446	44	11.2	3.13	1.33
63	78	27.3	13.6	2.00	3.81	125	479	44	11.1	3.13	1.40
64	76	26.0	14.3	2.14	4.00	111	479	43	11.1	3.13	1.33
65	99	27.5	12.5	1.66	2.50	61	396	56	13.0	3.85	2.18
66	79	26.0	13.3	2.00	2.90	111	462	45	11.5	3.13	1.51
67	99	30.0	17.6	1.80	2.66	71	429	56	11.4	3.13	1.96
68	88	28.6	14.3	2.00	2.66	61	462	50	12.7	3.33	2.06
69	78	27.3	15.8	2.30	3.00	80	479	44	10.9	3.33	1.40
Average	80	28.7	14.2	1.80	2.81	92	451	45	11.2	3.55	1.72
Maximum	104	36.0	21.4	2.72	4.00	133	495	59	14.3	5.00	2.45
Minimum	56	21.0	11.1	1.00	2.00	61	363	32	10.2	2.63	1.33

calcium content. That a high phosphorus intake will result in a reduced serum calcium value and an increased amount of serum phosphorus has been demonstrated by numerous investigators. Binger(93) in 1917-18 reported that intravenous injection of orthophosphoric acid and its sodium salts in dogs caused a diminution in the serum calcium and an increase in the serum phosphorus. Greenwald(94) did not agree with Binger and stated that the reduced calcium is not due to the phosphate ion but to changes in volume of blood, osmotic pressure, etc. This writer obtained similar calcium reduction by injecting sodium sulphate. Many investigators have since confirmed the work of Binger. In 1919, Meigs, Blatherwick, and Cary(95) reported experiments with cattle in which the addition of disodium phosphate to the ration increased the phosphorus concentration of the plasma and reduced the calcium content. Clark(96) demonstrated that animals on a low calcium intake may exhibit a decrease of 20 to 26 per cent in the calcium con-

TABLE 12.—*Blood chemical determinations on horses affected with osteomalacia.*

Animal No.	Percentage of hæmoglobin (calculated).	Milligrams per 100 cc blood.							Milligrams per 100 cc serum.		
		Non-protein nitrogen.	Urea nitrogen.	Creatinin.	Uric acid.	Sugar.	Chlorides as NaCl.	Iron.	Calcium.	Inorganic phosphorus.	Magnesium.
94	72	25.0	13.0	2.00	2.28	77	462	41	9.3	3.45	2.78
101	93	30.3	9.8	2.25	2.00	102	487	53	9.8	3.44	1.41
102	106	26.5	14.6	2.30	2.80	80	446	60	8.6	4.67	1.21
103	104	30.8	13.0	2.06	2.33	80	363	59	8.6	4.67	1.51
104	90	27.8	17.9	2.18	2.25	94	470	51	9.8	3.64	1.31
105	88	20.1	17.2	1.99	2.58	80	479	50	9.9	3.43	1.20
106	88	29.9	16.7	2.40	2.23	83	557	50	10.1	3.94	1.25
107	102	26.8	16.9	1.95	2.68	67	446	58	9.1	4.42	1.14
108	88	22.9	16.7	2.14	2.75	100	495	50	9.9	3.20	1.21
109	84	20.1	13.7	1.95	3.17	73	462	48	9.6	3.97	1.29
111	83	19.5	14.4	1.88	2.28	79	479	47	9.2	4.35	1.21
112	69	22.2	14.5	2.14	2.66	80	413	39	9.5	4.35	1.26
113	67	23.1	15.0	1.75	2.00	83	462	38	10.5	4.35	1.26
114	95	25.0	14.3	2.00	3.20	80	413	54	11.3	4.56	1.20
116	63	20.0	13.6	2.30	2.71	91	462	36	9.2	4.35	2.18
117	56	23.1	16.0	2.30	2.00	77	462	32	8.1	5.87	1.31
118	58	24.0	14.3	2.00	2.10	87	495	33	9.9	3.85	1.57
119	58	25.0	14.3	1.75	2.00	91	495	33	9.9	4.56	1.78
120	67	20.0	15.0	1.75	2.35	100	462	38	11.0	5.00	1.45
121	67	28.6	13.0	3.00	2.50	67	446	38	11.2	5.00	1.96
122	65	35.3	15.0	2.14	2.58	100	495	37	10.8	5.26	1.88
123	76	33.3	13.0	2.00	1.86	91	429	43	10.6	4.76	1.70
124	69	30.0	13.0	2.30	2.28	100	446	39	12.7	3.85	1.67
125	74	27.3	12.5	2.00	2.28	91	446	42	11.8	3.33	1.90
126	70	25.8	10.3	2.00	2.22	100	446	40	10.1	5.00	1.50
127	67	25.0	12.0	2.50	2.58	80	462	38	10.5	4.35	1.27
134	99	28.6	17.5	2.00	3.00	133	495	56	10.1	5.64	1.67
135	76	26.8	15.0	2.00	2.66	87	462	43	10.3	4.56	1.70
136	99	28.6	15.0	2.50	1.86	91	446	56	10.3	4.00	1.67
137	99	22.0	14.3	2.14	3.64	105	462	56	10.9	4.56	1.67
138	88	25.0	15.0	2.14	3.00	86	462	50	11.0	3.85	1.90
139	76	25.0	12.5	1.87	3.08	90	462	43	11.1	4.76	1.45
140	101	27.3	13.6	2.14	3.28	87	446	57	10.6	3.85	1.96
141	95	26.0	14.3	1.87	3.33	83	446	54	10.2	3.70	1.57
142	93	27.3	15.0	2.00	3.84	95	462	53	11.3	3.57	1.29
143	95	30.0	13.0	1.76	2.50	100	462	54	10.3	3.33	1.41
Average	81	25.9	14.3	2.09	2.57	88	460	45	10.2	4.26	1.55
Maximum	106	35.3	17.9	3.00	3.84	133	557	60	12.7	5.87	2.78
Minimum	56	19.5	9.8	1.75	1.86	67	363	32	8.1	3.20	1.20

tent of the blood after phosphate injection. Tisdall(97) demonstrated that a decided reduction of the serum calcium occurred and the serum phosphorus was increased greatly in dogs following intravenous injections of dibasic sodium phosphate. He

obtained similar results by intravenous injections of phosphoric acid. Greenwald,(98) however, did not agree with the statements of Tisdall. In 1922, Kramer and Howland(38) showed that if the phosphorus intake of young white rats remained at a normal level and the calcium intake was reduced, the serum calcium was reduced markedly while the serum phosphorus remained normal; an increase in dietary calcium resulted in increased serum calcium. Salvesen, Hastings, and McIntosh(99) demonstrated that large doses of phosphate administered to dogs over a period of one or two days produced violent symptoms similar to tetany, an increase in the serum phosphates, and a reduction of serum calcium. In 1927, Palmer and Eckles(100) reported that cattle fed on hay, which was abnormally low in phosphorus but adequate in calcium content, showed low plasma phosphorus and normal serum calcium. The addition of calcium carbonate did not affect the plasma phosphorus but increased the blood calcium while the addition of sodium phosphate increased the plasma phosphorus and reduced the blood calcium.

Ion products: blood of normal and osteomalacic horses.—In the following table the stoichiometric ion product $(Ca^{++})^3 \times (PO_4^{\equiv})^2$ has been calculated for all cases, both normal and osteomalacic, which have been studied in this series, consisting of sixty-nine of the former and thirty-six of the latter.

p_H determinations were made on eighteen normal cases and on twenty cases that were proven to be suffering with advanced osteomalacia. The method used was that described by Cullen,(101) modified by Hawkins,(102) and included, as modified, in the work by Hawk and Bergeim.(103) The average for each of the groups was 7.29. This figure was used in determining the factor (5.28) which was the basis for the calculation of the p product. The work of Holt(104) was used as a guide in the calculations.

$p(PO_4^{\equiv})$ was obtained from p(P) by the addition of the constant 5.28. The negative logarithm of the ion product, p product, was obtained by taking the sum of 3p(Ca) and $2p(PO_4^{\equiv})$. It has been found that in active rickets the p product is greater than 24.10, while in healing cases and normals the p product is less than 24.10.(104) It will be noted by reference to Tables 13 and 14 that our findings are not comparable to those of rickets, the p product being higher in the normals than in the osteomalacia cases. This would seem to be an almost certain indication of the withdrawal of calcium from the bones instead

TABLE 13.—*Stoichiometric ion product* $(Ca++)^3 \times (PO_4)^2$ *for normal horses.*

Ca.	[Ca ++].	Log. of [Ca ++].	p[Ca ++].	P.	[P].	Log. of [P].	p[P].	$p\left[PO_4^{\equiv\equiv}\right] = p[P] + 5.28.$	p[product].
10.3	2.58×10^{-3}	—3.41	2.59	2.94	0.95×10^{-3}	—2.96	3.04	8.32	24.41
11.6	2.90×10^{-3}	—3.46	2.54	3.45	1.11×10^{-3}	—3.05	2.95	8.23	24.08
12.1	3.03×10^{-3}	—3.48	2.52	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.06
11.5	2.88×10^{-3}	—3.46	2.54	2.94	0.95×10^{-3}	—2.96	3.04	8.32	24.26
10.5	2.62×10^{-3}	—3.42	2.58	3.23	1.04×10^{-3}	—3.02	2.98	8.26	24.26
12.3	3.10×10^{-3}	—3.49	2.51	3.57	1.15×10^{-3}	—3.06	2.94	8.22	23.97
11.3	2.82×10^{-3}	—3.45	2.55	2.86	0.92×10^{-3}	—2.97	3.03	8.31	24.27
11.1	2.78×10^{-3}	—3.44	2.56	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.18
12.8	3.20×10^{-3}	—3.51	2.49	3.70	1.19×10^{-3}	—3.08	2.92	8.20	23.87
12.3	3.10×10^{-3}	—3.49	2.51	3.85	1.24×10^{-3}	—3.09	2.91	8.19	23.91
13.2	3.30×10^{-3}	—3.52	2.48	3.33	1.07×10^{-3}	—3.03	2.97	8.25	23.94
12.9	3.23×10^{-3}	—3.51	2.49	4.17	1.35×10^{-3}	—3.13	2.87	8.15	23.77
13.5	3.38×10^{-3}	—3.53	2.47	3.23	1.04×10^{-3}	—3.02	2.97	8.25	23.91
13.7	3.43×10^{-3}	—3.54	2.46	3.85	1.24×10^{-3}	—3.09	2.91	8.19	23.76
14.0	3.50×10^{-3}	—3.54	2.46	3.13	1.01×10^{-3}	—3.00	3.00	8.28	23.94
14.3	3.58×10^{-3}	—3.55	2.45	4.00	1.29×10^{-3}	—3.11	2.89	8.17	23.69
10.5	2.62×10^{-3}	—3.42	2.58	3.70	1.19×10^{-3}	—3.08	2.92	8.20	24.14
10.9	2.78×10^{-3}	—3.44	2.56	4.17	1.35×10^{-3}	—3.13	2.87	8.15	23.98
11.1	2.78×10^{-3}	—3.44	2.56	3.70	1.19×10^{-3}	—3.08	2.92	8.20	24.08
11.3	2.82×10^{-3}	—3.45	2.55	3.03	0.98×10^{-3}	—2.99	3.01	8.29	24.23
10.2	2.55×10^{-3}	—3.41	2.59	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.15
11.1	2.78×10^{-3}	—3.44	2.56	4.00	1.29×10^{-3}	—3.11	2.89	8.17	24.02
11.3	2.82×10^{-3}	—3.45	2.55	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.15
10.7	2.68×10^{-3}	—3.43	2.57	4.00	1.29×10^{-3}	—3.11	2.89	8.17	24.05
11.1	2.78×10^{-3}	—3.44	2.56	4.17	1.35×10^{-3}	—3.13	2.87	8.15	23.98
10.9	2.73×10^{-3}	—3.44	2.56	4.00	1.29×10^{-3}	—3.11	2.89	8.17	24.02
11.8	2.95×10^{-3}	—3.47	2.53	3.70	1.19×10^{-3}	—3.08	2.92	8.20	23.99
11.7	2.93×10^{-3}	—3.47	2.53	4.35	1.40×10^{-3}	—3.15	2.85	8.13	23.85
12.1	3.03×10^{-3}	—3.48	2.52	4.00	1.29×10^{-3}	—3.11	2.89	8.17	23.90
11.5	2.88×10^{-3}	—3.46	2.54	4.17	1.35×10^{-3}	—3.13	2.87	8.15	23.92
10.2	2.55×10^{-3}	—3.41	2.59	2.63	0.85×10^{-3}	—2.93	3.07	8.35	24.47
10.6	2.65×10^{-3}	—3.42	2.58	3.70	1.19×10^{-3}	—3.08	2.92	8.20	24.14
11.7	2.93×10^{-3}	—3.47	2.53	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.09
12.6	3.15×10^{-3}	—3.50	2.50	3.85	1.24×10^{-3}	—3.09	2.91	8.19	23.88
11.2	2.80×10^{-3}	—3.45	2.55	5.00	1.61×10^{-3}	—3.21	2.79	8.07	23.79
11.0	2.75×10^{-3}	—3.40	2.60	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.30
11.6	2.90×10^{-3}	—3.46	2.54	3.57	1.15×10^{-3}	—3.06	2.94	8.22	24.06
10.7	2.68×10^{-3}	—3.43	2.57	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.21
10.5	2.62×10^{-3}	—3.42	2.58	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.12
11.8	2.95×10^{-3}	—3.47	2.53	3.85	1.24×10^{-3}	—3.09	2.91	8.19	23.97
11.9	2.98×10^{-3}	—3.47	2.53	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.15
10.6	2.65×10^{-3}	—3.42	2.58	3.57	1.15×10^{-3}	—3.06	2.94	8.22	24.18
10.8	2.70×10^{-3}	—3.43	2.57	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.09
10.5	2.62×10^{-3}	—3.42	2.58	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.30
11.2	2.80×10^{-3}	—3.45	2.55	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.21
10.8	2.70×10^{-3}	—3.43	2.57	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.27
12.9	3.23×10^{-3}	—3.51	2.49	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.03
10.2	2.55×10^{-3}	—3.41	2.59	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.33
10.2	2.55×10^{-3}	—3.41	2.59	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.15

TABLE 13.—*Stoichiometric ion product* $(Ca++)^3 \times (PO_4)^2$ *for normal horses*—Continued.

Ca.	[Ca ++].	Log. of [Ca ++].	p[Ca ++].	P.	[P].	Log. of [P].	p[P].	$p\left[PO_4^{\equiv}\right] + 5.28$.	p[product].
10.2	2.55×10^{-3}	—3.41	2.59	4.56	1.47×10^{-3}	—3.17	2.83	8.11	23.99
10.9	2.73×10^{-3}	—3.44	2.56	4.00	1.29×10^{-3}	—3.11	2.89	8.17	24.02
10.6	2.65×10^{-3}	—3.42	2.58	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.12
10.2	2.55×10^{-3}	—3.41	2.59	3.45	1.11×10^{-3}	—3.05	2.95	8.23	24.23
11.0	2.75×10^{-3}	—3.40	2.60	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.18
10.6	2.65×10^{-3}	—3.42	2.58	3.45	1.11×10^{-3}	—3.05	2.95	8.23	24.20
10.8	2.70×10^{-3}	—3.43	2.57	4.17	1.35×10^{-3}	—3.13	2.87	8.15	24.01
10.8	2.70×10^{-3}	—3.43	2.57	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.27
10.9	2.73×10^{-3}	—3.44	2.56	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.06
11.0	2.75×10^{-3}	—3.40	2.60	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.36
11.2	2.80×10^{-3}	—3.45	2.55	3.03	0.98×10^{-3}	—2.99	3.01	8.29	24.23
11.0	2.75×10^{-3}	—3.40	2.60	3.03	0.98×10^{-3}	—2.99	3.01	8.29	24.38
11.2	2.80×10^{-3}	—3.45	2.55	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.21
11.1	2.78×10^{-3}	—3.44	2.56	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.24
11.1	2.78×10^{-3}	—3.44	2.56	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.24
13.0	3.25×10^{-3}	—3.51	2.49	3.85	1.24×10^{-3}	—3.09	2.91	8.19	23.85
11.5	2.88×10^{-3}	—3.46	2.54	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.18
11.4	2.85×10^{-3}	—3.46	2.54	3.13	1.01×10^{-3}	—3.00	3.00	8.28	24.18
12.7	3.20×10^{-3}	—3.51	2.49	3.33	1.07×10^{-3}	—3.03	2.97	8.25	23.97
10.9	2.73×10^{-3}	—3.44	2.56	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.18
AVERAGE									
11.2	2.85×10^{-3}	—3.45	2.55	3.55	1.15×10^{-3}	—3.05	2.95	8.23	24.87

of a mere failure to deposit or to act as an indicator of quiescence. This is supported by our findings that the phosphorus ratio is high in these animals and that the phosphate intake of animals subsisting on Philippine products is extraordinarily high and requires the addition of calcium to the ration to balance the excessive phosphorus taken in the feed.

Bone.—The study of the chemical changes occurring in the bones of animals affected with osteomalacia has held the attention of many investigators. In 1905, Friedberger and Fröhner(5) reported that the specific gravity of affected bones is diminished, the lime content reduced more than one-half, and the water content increased. Mohler(1) reported (1906) that chemical analysis of bones from affected animals showed a reduction of fat, phosphoric acid, lime, and soda and an increase in organic matter and silicic acid. McCrudden(105) in 1906–07 compared the analysis of the ribs from two horses affected with

TABLE 14.—*Stoichiometric ion product* $(Ca^{++})^3 \times (PO_4^{\equiv})^2$ *for osteomalacic horses.*

Ca.	[Ca ++].	Log. of [Ca ++].	p[Ca ++].	P.	[P].	Log. of [P].	p[P].	$p[PO_4^{\equiv}]$ +5.28.	p[product].
9.3	2.33×10^{-3}	—3.37	2.63	3.45	1.11×10^{-3}	—3.05	2.95	8.23	24.35
9.8	2.45×10^{-3}	—3.39	2.61	3.44	1.11×10^{-3}	—3.05	2.95	8.23	24.29
8.6	2.15×10^{-3}	—3.33	2.67	4.67	1.51×10^{-3}	—3.18	2.82	8.10	24.21
8.6	2.15×10^{-3}	—3.33	2.67	4.67	1.51×10^{-3}	—3.18	2.82	8.10	24.21
9.8	2.45×10^{-3}	—3.39	2.61	3.64	1.17×10^{-3}	—3.07	2.93	8.21	24.25
9.9	2.48×10^{-3}	—3.39	2.61	3.43	1.11×10^{-3}	—3.05	2.95	8.23	24.29
10.1	2.52×10^{-3}	—3.40	2.60	3.94	1.27×10^{-3}	—3.10	2.90	8.18	24.16
9.1	2.28×10^{-3}	—3.36	2.64	4.42	1.43×10^{-3}	—3.15	2.85	8.13	24.18
9.9	2.48×10^{-3}	—3.39	2.61	3.20	1.03×10^{-3}	—3.01	2.99	8.27	24.37
9.6	2.40×10^{-3}	—3.38	2.62	3.97	1.28×10^{-3}	—3.11	2.89	8.17	24.20
9.2	2.30×10^{-3}	—3.36	2.64	4.35	1.40×10^{-3}	—3.15	2.85	8.13	24.18
9.5	2.38×10^{-3}	—3.38	2.62	4.35	1.40×10^{-3}	—3.15	2.85	8.13	24.12
10.5	2.62×10^{-3}	—3.42	2.58	4.35	1.40×10^{-3}	—3.15	2.85	8.13	24.00
11.3	2.82×10^{-3}	—3.45	2.55	4.56	1.47×10^{-3}	—3.17	2.83	8.11	23.87
9.2	2.30×10^{-3}	—3.36	2.64	4.35	1.40×10^{-3}	—3.15	2.85	8.13	24.18
8.1	2.02×10^{-3}	—3.31	2.69	5.87	1.98×10^{-3}	—3.30	2.70	7.98	24.03
9.9	2.48×10^{-3}	—3.39	2.61	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.21
9.9	2.48×10^{-3}	—3.39	2.61	4.56	1.47×10^{-3}	—3.17	2.83	8.11	24.05
11.0	2.75×10^{-3}	—3.40	2.60	5.00	1.61×10^{-3}	—3.21	2.79	8.07	23.94
11.2	2.80×10^{-3}	—3.45	2.55	5.00	1.61×10^{-3}	—3.21	2.79	8.07	23.79
10.8	2.70×10^{-3}	—3.43	2.57	5.26	1.70×10^{-3}	—3.23	2.77	8.05	23.81
10.6	2.65×10^{-3}	—3.42	2.58	4.76	1.54×10^{-3}	—3.19	2.81	8.09	23.92
12.7	3.20×10^{-3}	—3.51	2.49	3.85	1.24×10^{-3}	—3.09	2.91	8.19	23.85
11.8	2.95×10^{-3}	—3.47	2.53	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.09
10.1	2.53×10^{-3}	—3.40	2.60	5.00	1.61×10^{-3}	—3.21	2.79	8.07	23.94
10.5	2.62×10^{-3}	—3.42	2.58	4.35	1.40×10^{-3}	—3.15	2.85	8.13	24.00
10.1	2.53×10^{-3}	—3.40	2.60	5.64	1.82×10^{-3}	—3.26	2.74	8.02	23.84
10.3	2.58×10^{-3}	—3.41	2.59	4.56	1.47×10^{-3}	—3.17	2.83	8.11	23.99
10.3	2.58×10^{-3}	—3.41	2.59	4.00	1.29×10^{-3}	—3.11	2.89	8.17	24.11
10.9	2.73×10^{-3}	—3.44	2.56	4.56	1.47×10^{-3}	—3.17	2.83	8.11	23.90
11.0	2.75×10^{-3}	—3.40	2.60	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.18
11.1	2.78×10^{-3}	—3.44	2.56	4.76	1.54×10^{-3}	—3.19	2.81	8.09	23.86
10.6	2.65×10^{-3}	—3.42	2.58	3.85	1.24×10^{-3}	—3.09	2.91	8.19	24.12
10.2	2.55×10^{-3}	—3.41	2.59	3.70	1.19×10^{-3}	—3.08	2.92	8.20	24.17
11.3	2.82×10^{-3}	—3.45	2.55	3.57	1.15×10^{-3}	—3.06	2.94	8.22	24.09
10.8	2.70×10^{-3}	—3.43	2.57	3.33	1.07×10^{-3}	—3.03	2.97	8.25	24.21
AVERAGE									
10.2	2.55×10^{-3}	—3.40	2.60	4.26	1.38×10^{-3}	—3.14	2.87	8.15	24.08

osteomalacia with the bones from two normal horses. He reported a decrease in calcium content and an increase in the magnesium, stating that these results confirm the analyses of Roloff (1866) and Chabrie (1895). He also reported that in

osteomalacia there is an increase in sulphur and a decrease in phosphorus and inorganic material. In 1907, Ingle(16) reported the analysis of bones from horses, mules, and donkeys affected with osteomalacia and compared these results with analyses of normal bone. He found that normal bone contained larger proportions of ash, lime, and phosphoric acid, and that there was little difference in the composition of bones from affected animals, though much difference in the amount. Ramsey(54) showed that samples of bones of animals reared in sections where osteomalacia was prevalent, contained less lime and phosphoric acid than the bones of animals reared in sections where the disease was not prevalent.

Forbes(3) fed swine a ration in which there was insufficient mineral bases to neutralize the mineral acids present and found that a reduction in the total ash of the bone had occurred, as well as a loss in breaking strength. This writer states that Weiska observed a decrease in the ash of bones from rabbits and sheep that had been fed monosodium phosphate. Bohstadt, Bethke, Edgington, and Robison(106) showed that the average ash content of bones of swine was increased approximately 10 per cent when calcium carbonate was added to a basal ration. These writers showed that a positive correlation existed between the ash content and the breaking strength, and that while the percentage of total ash varied decidedly, the ratio of calcium to phosphorus to magnesium in the ash remained nearly constant.

In the present investigation determinations were made of the ash and chemical composition of bones from affected and normal animals. The tensile strength was determined also. In each instance the left metacarpal bone was used for the determinations. Clinical examination of the animal and X-ray examination of the metacarpal bone were made on each case used in these tests. The total ash determination on eight normal animals ranged from 61.50 per cent to 65.11 per cent with an average of 63 per cent ash. This determination on a total of thirty cases of osteomalacia showed a range of 56.41 per cent to 61.46 per cent with an average of 60.76 per cent ash. Based on the X-ray examination, the bones were grouped according to the degree of rarefaction present. It was found that bones showing the greatest rarefaction, showed the greatest reduction in the percentage of ash. Table 15 shows in detail the ash determinations, while fig. 12 presents these data graphically.

TABLE 15.—*Ash content of normal and osteomalacic bones.*

	Normal.	Osteomalacia.		
		Mild.[a]	Moderate.[a]	Severe.[a]
	Per cent.	*Per cent.*	*Per cent.*	*Per cent.*
	61.66	61.75	59.23	60.37
	65.11	63.25	62.22	59.94
	62.29	59.32	60.67	58.67
	62.02	60.14	59.96	56.66
	61.50	63.88	60.54	61.62
	63.07	60.70	62.92	----------
	64.28	61.67	62.63	----------
	64.04	58.91	61.58	----------
	----------	63.12	59.50	----------
	----------	61.18	60.27	----------
	----------	62.59	60.72	----------
	----------	60.21	56.41	----------
	----------	62.26	----------	----------
Average	63.00	61.46	60.55	59.45
Maximum	65.11	63.88	62.92	61.62
Minimum	61.50	58.91	56.41	56.66

[a] As indicated by X-ray examination.

Average analysis of the ash from bones of eight normal horses showed, calcium, 37.35 per cent; phosphorus, 18.32 per cent; and magnesium, 0.13 per cent. The results on thirty osteomalacic cases were, calcium, 37.54 per cent; phosphorus, 18.23 per cent; and magnesium, 0.19 per cent.

The results of the above analyses confirm the statement that in osteomalacia there is a reduction of the total mineral content of the bone. The percentage of calcium and phosphorus in the bone ash is approximately the same for affected as for normal bone; the magnesium content of ash from osteomalacic bone shows a marked increase.

Comparative tests were conducted to determine the tensile strength of normal and osteomalacic bones. The left metacarpal bone was used in all instances, and the clinical diagnosis of osteomalacia was confirmed by X-ray examination of the bone. In order to correlate variable conditions present in the different specimens—such as, difference in the width and length of the shaft of the bone, thickness of cortex, and extent of medulla, an arbitrary formula was devised and applied in all cases. In devising this formula, the bone at the fracture point (middle of shaft) was considered as being elliptical and the medulla at this point, as circular in shape. Measurements were then taken

at the fracture point, the total cross-sectional area calculated and from this was deducted the area occupied by the medullary canal; that is, the cross-sectional area of the cortex at the point of fracture was estimated. The average area for fourteen normal and twenty-six osteomalacic bones was 8.44 square centimeters. The total length of the shaft constituted the span between two stationary supports. Mechanical weight was applied midway between the supports, using a Tinus Olsen (Philadelphia, Pennsylvania) machine devised for testing the tensile strength of materials. It was found that the average span for all bones tested was 15.3 centimeters. The arbitrary formula used to correlate the variable factors is as follows:

$$\frac{\frac{a \times b}{B} Y}{y} = A$$

a = kilograms weight to produce fracture.
b = average span of 40 metacarpal bones (15.3 centimeters).
B = span of specimen.
Y = cross-sectional area of cortex at point of fracture of specimen.
y = average cross-sectional area of 40 metacarpal bones (8.44 square centimeters).
A = corrected weight required to fracture specimen.

Using this formula to correct the normal variations found in the metacarpal bones of various individuals, it was found that on 14 normal cases the average weight required was 1,669 kilos, the maximum being 3,615 and the minimum 746. The average age of these horses was 13.9 years. Applying the same calculations and technic to 26 metacarpal bones from osteomalacic horses it was found that the average weight required was 1,145 kilos, with a maximum of 1,817 and a minimum of 620. The average age of the osteomalacia cases was 13.5 years.

In this experiment on the tensile strength of bones it was demonstrated that the metacarpals from horses affected with osteomalacia required an average of approximately 31 per cent less weight to cause fracture than did normal bones under the same conditions.

CLINICAL ASPECTS

General.—During the present investigation it was noted that the individual resistance to osteomalacia was extremely variable,

some animals developing the condition within six months after arrival in the Philippines while others, maintained under similar conditions for a period of more than five years, did not develop the disease. As shown elsewhere in this article, mules exhibit a far greater resistance to the condition than do horses. While native animals in some sections of the Philippines show a remarkable resistance to osteomalacia, as was demonstrated by an examination made in Cebu, Cebu, of 275 native ponies of which only two showed clinical evidence of the disease, in other sections the disease is prevalent and constitutes an important economic factor in the development of the live-stock industry.

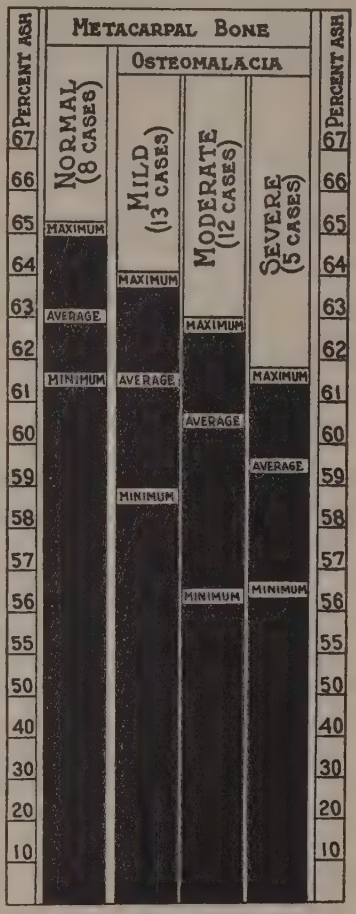

FIG. 12. Ash content of normal and osteomalacic bones.

The rapidity with which the disease developed in Army animals in the Philippines is extremely variable. The shortest period of service before the condition appeared was approximately five months. This case occurred in an 8-year-old horse which arrived in Manila October 18, 1930, was shipped to Fort Stotsenburg December 19, 1930, and was diagnosed as osteomalacia March 7, 1931. The animal was normal on arrival. As compared with this short developmental period, 78 cases occurred during 1930 and 1931, in animals that were brought to Fort Stotsenburg in 1924 or earlier; the date of arrival of this group in the Philippines is not recorded. Out of a shipment of 72 horses and 97 mules arriving in the Islands October 18, 1930, and shipped to Fort Stotsenburg December 19, 1930, two horses, or 2.8 per cent, de-

veloped the disease within six months after arrival in the Philippines; of the 97 mules in this shipment, no cases were recorded up to January 1, 1932. The incidence rate of the disease in animals having approximately one year of service at Fort Stotsenburg is shown in Table 16.

TABLE 16.—*Incidence of osteomalacia among animals having approximately one year of service at Fort Stotsenburg.*

Arrived.		Animals in shipment.			Osteomalacia.					
					Horses.		Mules.		Total.	
Philippine Islands.	Fort Stotsenburg.	Horses.	Mules.	Total.	Cases.	Per cent.	Cases.	Per cent.	Cases.	Per cent.
Dec. 14, 1929	Feb. 13, 1930	97	145	242	10	10.3	5	3.4	15	6.2
July 10, 1930	Aug. 9, 1930	31	90	121	3	9.7	1	1.1	4	3.4
Total		128	235	363	13	10.1	6	2.5	19	5.2

All cases of osteomalacia noted in the above table, occurred between November 12, 1930, and June 29, 1931. It will be noted that 13 cases, or approximately 10 per cent, of the 128 horses developed the condition in about one year while six, or approximately 2.5 per cent, of the 235 mules developed the disease in the same period.

Table 17 shows the percentage of horses and mules that developed the condition at Fort Stotsenburg during the periods indicated. All animals were shipped to the Philippines in recent years.

TABLE 17.—*Duration of service at Fort Stotsenburg before osteomalacia developed.*

Length of service (approximate) in Philippine Islands.	Animals in shipment.		Osteomalacia.	
	Horses.	Mules.	Horses.	Mules.
Years.			*Per cent.*	*Per cent.*
0.6	72	97	2.8	0
1	31	90	9.7	1.1
1.5	97	145	10.3	3.4
2		159		5.0
2.25	39	24	35.9	0
2.5		125		4.8
3.5	24	90	38.0	10.0
4	88	4	15.9	25.0

Symptomatology.—The symptoms of osteomalacia in animals vary with the species and depend somewhat on the natural position assumed by the animal. Thus, in monkeys, thoracic and pelvic deformity is relatively commoner than in carnivores and rodents due to the fact that the former tend to sit in one position during the developmental period of the disease whereas the latter tend to lie in one position.(24)

The incipient stage in Herbivora shows no recognizable change and the earliest symptoms are changes in the activity of the animal, there being a marked lessening of voluntary movement. Some writers state that perverted appetite is an early symptom but this has not been noted during the present investigation. The disease develops gradually in horses. An intermittent and shifting lameness with no obvious cause, usually manifested by a characteristic shortening of the stride of either the front or hind legs or both, is an early symptom. This lameness is increased by weight-bearing; it may disappear on rest only to recur in some other joint. While standing, the animal usually shifts its weight, thus resting affected parts. The shoulder and hip joints are involved commonly. As the disease progresses, the lameness becomes more marked, the stride exceedingly shortened and a characteristic "tucked-up" appearance of the flanks is present, particularly when the animal is required to trot.

Some cases exhibit a bilateral enlargement of the facial bones at an early stage, while others do not present this symptom even when the disease is well developed. Approximately 5 per cent of the cases examined during the present investigation exhibited proliferative changes of the facial bones. The nasal passages were narrowed when the facial bones were affected. Bony enlargements of the mandible were found in about 98 per cent of the cases examined and this feature provides a valuable, early diagnostic sign. This proliferative change is noted along that portion of the ramus of the mandible in which the lower molar teeth are embedded and is detected readily by palpation of the superior, lateral border of the mandible adjacent to the molar teeth. In the normal animal, this mandibular border is smooth and almost perpendicular to the molar teeth; any enlargement, thickening, or ridge at this point should be viewed as suspicious of osteomalacia. This proliferative change is bilateral (see Plate 10). Several cases were seen in which this proliferative change of the mandible was the only symptom

apparent. Some cases show periarticular exostoses. When the disease is fairly well advanced lordosis may be present. A depression of the ribs sometimes occurs that causes a deformity of the chest, as a consequence of which the shoulder joints appear to be displaced forward causing a sunken appearance of the sternum (see Plates 1 and 3). Fractures and torn ligaments may occur but are not frequent, because of the fact that affected animals are destroyed usually before the condition has advanced to the stage where fractures might occur. Fractures of the cervical and lumbar vertebræ and of the metatarsal bones, and torn ligaments of the hock joint occurred among our experimental animals. Enlargement of the ribs at the costochondral junction was noted in several cases.

In the early stage of the disease the animal appears to be well nourished but later becomes poor in flesh, the hair coat becomes rough, anorexia develops, and the animal experiences difficulty in rising. The temperature remains normal. Several cases observed showed an intermittent leucocytosis, the white cell count reaching 17,000 in some.

The age factor has been discussed elsewhere.

DIAGNOSIS

Clinical.—During the early stage of the disease, marked by an intermittent, shifting lameness only, diagnosis is difficult, particularly when exostoses or other pathological conditions are present concurrently. The lameness is diagnosed frequently as spavin or ring bone lameness, arthritis, bursitis, rheumatism, etc. The disease may be suspected when lameness, shifting and intermittent in character and without apparent cause, is present. In the majority of cases the first bone deformity to appear is the characteristic rounding of the superior border of the mandible lateral to the molar teeth. It is believed that this deformity is pathognomic of osteomalacia in the Equidæ. In later stages of the disease, the stride is characteristic, it being markedly shortened and choppy.

X-ray.—We were unable to find references in available literature referring to X-ray findings in equine osteomalacia. In routine X-ray examinations, the metacarpal bones were selected, primarily because of the ease with which X-ray pictures of this bone could be made and it is believed that these bones show all pathological details that might be found in other bones. The technic follows:

Restraint of subject.—Numerous attempts were made to obtain X-ray pictures of the metacarpal bone with the animal in

the standing position. These were unsatisfactory, as the slightest movement destroyed the detail of the picture and it was found necessary to use an equine operating table (H. & D., Kyle) in order to arrest completely all movement of the part.

X-ray equipment.—A portable, bedside unit (Waite Dartledt Co., New York) equipped with Coolidge, radiator-type tube was used. Cassettes fitted with double intensifying screens completed the equipment. A constant distance of 20 inches and a milliamperage of 8 was used. The time of exposure varied from 5 to 8 seconds depending upon the thickness of the metacarpal bone.

X-ray findings.—No enlargement was evident in any of the metacarpal bones examined. The most striking feature was a haziness of outline both in the cortical and medullary portions of the bone. There was an indistinct outline between the cortical and medullary portions throughout the length of the bone and an irregular thinning of the cortical portion with a replacement by the medullary cavity. Longitudinal striations, indicating a decalcification process, were present in the compact portion of the metacarpal bone in all clinical cases. Apparently normal animals of advanced age may show some striations of the cortex (senile osteoporosis). The inner outline of the cortex was irregular, showing that the destructive process of compact bone was operating from within outward. In many cases there was a very definite roughening at the tendinous attachment of the extensor carpi radialis muscle. The sesamoid bones show usually a rarefaction and striation equally as extensive as that found in the metacarpal bones. It is believed that the X-ray affords an accurate means of diagnosing osteomalacia. Plates 23 and 24 illustrate X-ray findings in normal bone, mild, moderate, and severe osteomalacia. Negatives have not been retouched for any X-ray photographs used in this article.

Chemical.—It has been shown that in equine osteomalacia there is a decrease in the serum calcium and an increase in the inorganic phosphorus of the serum. These changes are not constant throughout the course of the disease. A series of daily determinations of serum calcium and phosphorus was made on a number of typical cases of the disease and it was found that on some days these elements were present in the blood in normal amounts. For the above reasons, a single determination of the serum calcium and phosphorus is likely to be inconclusive. It is believed that a series of daily determinations would be of diagnostic value.

Course and prognosis.—Equine osteomalacia is usually chronic in character and, with temporary improvements and relapses, may extend over a period of several years. The majority of cases develop slowly and the animal remains semiserviceable for a comparatively long period of time. Some cases, however, develop within a short time and the animal becomes unserviceable within six to eight months. Hard work and adverse conditions of animal management seem to hasten the course of the disease. Individual resistance of the animal, pregnancy, and gestation have some influence on the course of the disease undoubtedly. Spontaneous recovery may occur although this is unusual.

Fig. 13 shows the disposition of 340 cases of the disease which occurred at Fort Stotsenburg during the period July 1, 1930, to July 1, 1931.

With the application of proper corrective measures to the ration, many cases recovered completely and were restored to full military duty. Several experimental cases were returned to duty after two months treatment, and at the completion of the experiment, nine months later, these animals were still doing duty, having suffered no relapses during this period. While the bony proliferations of the head persisted invariably, several instances were noted in which these enlargements subsided in a small degree.

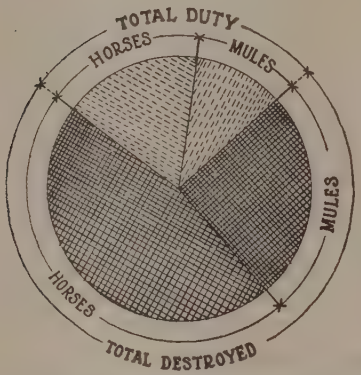

FIG. 13. Disposition of osteomalacia cases at Fort Stotsenburg from July 1, 1930, to July 1, 1931 (340 cases).

Records show that 224 cases were destroyed. The ages of these animals are shown in Table 18. Fig. 14 shows the number of animals in age groups of two years each and the percentage of cases in each group.

PREVENTION AND TREATMENT

At this time we wish to emphasize the fact that all statements made heretofore and to be made hereafter apply only to the osteomalacia in horses with which we have been dealing in the Philippines. We are not prepared to say whether these

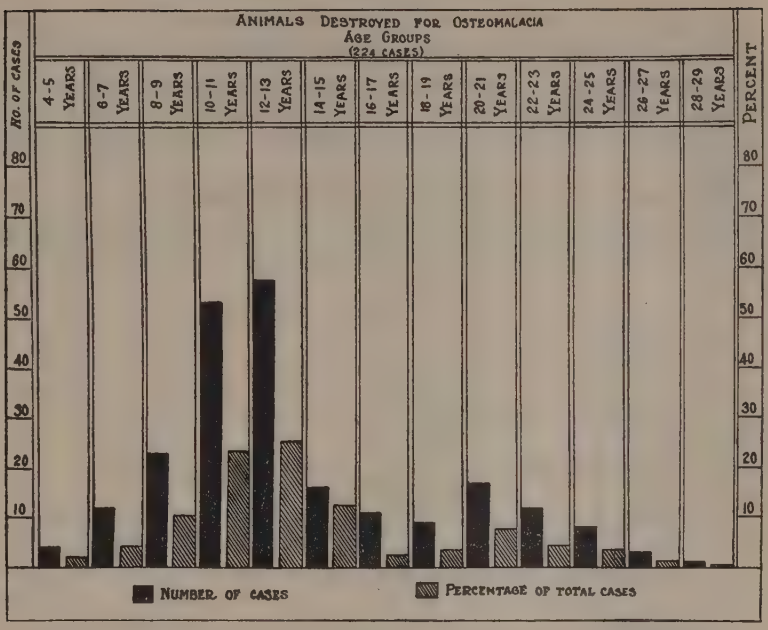

FIG. 14. Age groups of animals destroyed for osteomalacia at Fort Stotsenburg.

TABLE 18.—*Ages of animals with osteomalacia destroyed at Fort Stotsenburg from July 1, 1930, to July 1, 1931 (224 cases).*

Age in years.	Horses.	Mules.	Age in years.	Horses.	Mules.
5	3	2	17		1
6	4	1	19	5	4
7	1	6	20	5	1
8	5		21	8	3
9	16	2	22	2	2
10	17	6	23	5	3
11	28	3	24	6	2
12	23	10	26	1	2
13	16	4	29		1
14	9	1			
15	4	2			
16	3	2	Totals	166	58

statements will apply to osteomalacia as found in the same animals in other locations or in other animals.

The prevention of equine osteomalacia in the Philippines depends entirely on the feeding of a ration properly balanced, with special reference to its mineral components. We have

been unable to demonstrate any vitamin deficiency. The ration should be balanced in such a manner that the calcium oxide-phosphorus pentoxide intake ratio is between 1 : 1 and 1 : 1.8. This balance may be obtained by the addition of calcium to the ration direct or through its addition in the natural water supply.

Reference to fig. 15 will show that osteomalacia did not occur at Fort Mills (Corregidor) where the water supply contained a relatively large proportion of calcium and that the case rate

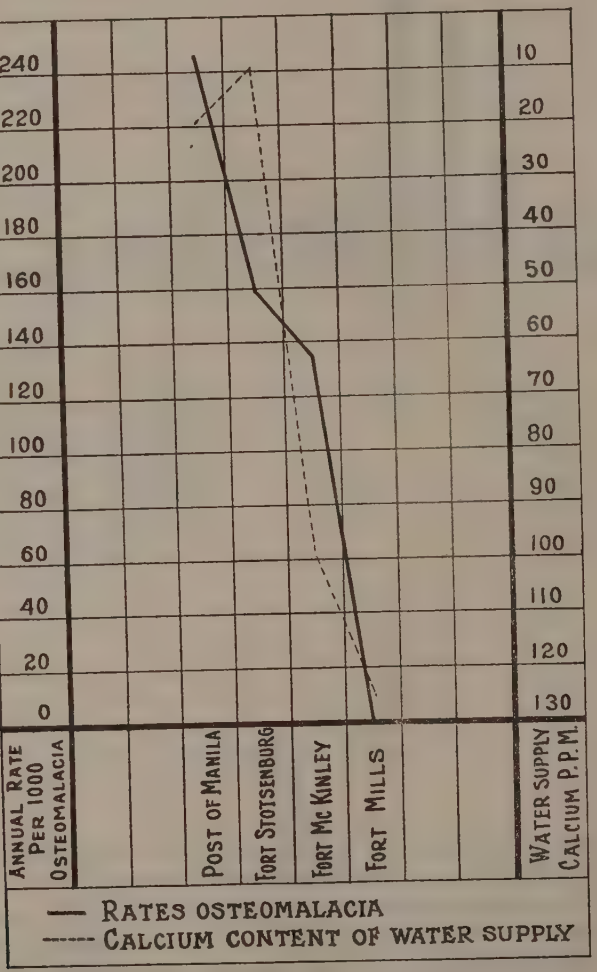

FIG. 15. Comparison of osteomalacia incidence and calcium in water supply.

increased at other posts approximately in proportion to the drop in the calcium content. It must be remembered in this connection that the conditions were otherwise identical at all posts. This includes ration, animal management and care, working conditions, etc.

Fig. 16 shows graphically the total admission rate (less infectious diseases) and admission rate for osteomalacia prior to and subsequent to the institution of a new ration wherein the calcium oxide-phosphorus pentoxide intake ratio was balanced at a figure of approximately 1 : 1.

It has been shown that there was a sharp increase in the admission rate for osteomalacia for the year 1930. It is our belief that this is in part artificial due to the fact that a complete survey of all animals on the post had not been held prior to 1930 and the additional fact that a certain number of osteomalacia cases were diagnosed probably as other conditions prior to this time. It has been shown that since the general admission rate for all conditions, other than infectious diseases, was high prior to 1930, osteomalacia cases probably existed in great numbers prior to that year. It will be noted that, subsequent to the institution of the new ration in June, 1931, there was a prompt and marked drop in the osteomalacia cases reported and at the same time a drop in the general admission rate for all causes other than infectious disease. This but supports our contention that osteomalacia has been included, for the most part, in the guise of other disease entities prior to 1930.

No medicinal treatment was employed in any case except for symptomatic conditions, the results obtained following the alteration in the ration only.

COMMENTS AND CONCLUSIONS

1. A comprehensive review of available literature has been undertaken and pertinent excerpts included herewith. History and occurrence of the disease as a world-wide entity has been commented upon, while in the Philippines the same subjects have been discussed in detail. Similar diseases and those diseases for which osteomalacia has been mistaken have received their share of attention.

2. The great economic importance of osteomalacia to the United States Army in the Philippines and to a lesser extent to the civilian population cannot be overemphasized. In so far as the Army is concerned it has resulted in serious interference with

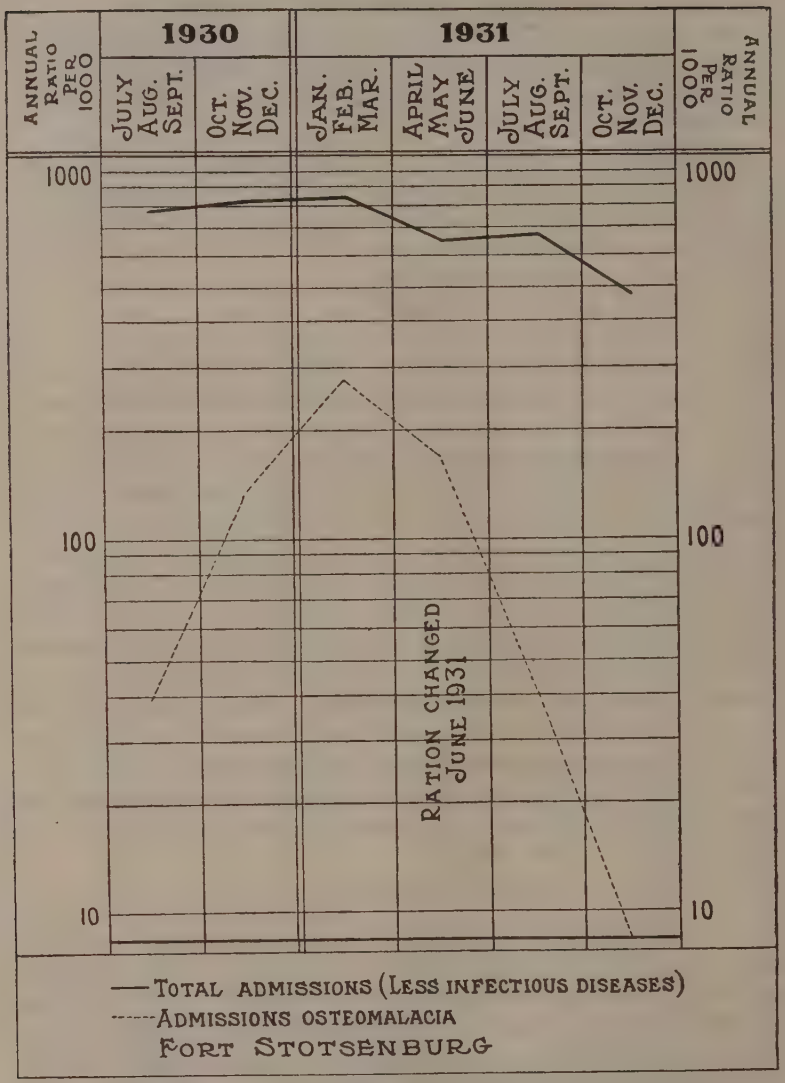

FIG. 16. Total admission rates and rates for osteomalacia at Fort Stotsenburg.

transportation and a monetary loss that cannot be calculated accurately but is high undoubtedly.

3. Native forage cannot be utilized, unsupplemented by mineral additions, but can be taken advantage of provided the calcium-phosphorus ratio is adjusted properly, either by increments of calcium from the natural water supply or by the addition direct to the ration. This latter is required at all

Army posts in the Philippines except Fort Mills (Corregidor). The fact that native forage can be used by the Army in the Philippines results in an advantage to the local population in that they are enabled to dispose of a large quantity of forage for the feeding of Army animals; the United States Government is not dependent entirely on imported forage, is saved the cost of shipment, and the reduced price of certain forage products obtainable in the Philippines is an additional advantage.

4. Diseases affecting the bones and organs of locomotion were found to be more prevalent in Army animals in the Philippines than in like groups in the United States.

5. Osteomalacia affects mules as well as horses, but the former exhibit far more resistance. The disease affects animals of all ages but particularly those animals in the age group between six to thirteen years. No evidence was found indicating that sex played any part in the development of the disease. The effects of pregnancy could not be studied due to the fact that breeding is not an activity of the Army in the Philippines.

6. The cause of the osteomalacia exhibited by Army animals in the Philippines appears to be due solely to a mineral imbalance. There was no evidence that disturbance of internal secretion, parasitic infestation, heredity, infection, or vitamin deficiency played any part in the production of the disease. It has been found that in the prodromal stage as well as in the stage of active manifestation of osteomalacia there is a reduction in serum calcium and an increase in inorganic phosphorus and that this relationship may be altered by the administration of an increased ratio of calcium resulting in an improvement of the condition. It seems fair to conclude that a reduction in the phosphate intake would accomplish the same results since an increase in the phosphorus intake ratio resulted in an increase in the severity of the disease.

7. Macro- and micropathology have been discussed in detail. It has been found that the condition dealt with is definitely osteomalacia, and not the so-called osteoporosis, by reason of the fact that there is a definite replacement of the mineral salts in the bones by osteoid tissue.

8. Average blood-chemical findings on nonprotein nitrogen, urea nitrogen, creatinin, uric acid, and sugar on sixty-nine normal horses have been added to a rather meager literature on the subject. The hæmoglobin as calculated from the iron content, iron, chlorides as sodium chloride, calcium, inorganic phos-

phorus, and magnesium have been determined for the same number of normal animals. The latter determinations form the first comprehensive work on the subject.

The same determinations have been made on thirty-six cases of osteomalacia. The only noteworthy difference between the normal and the osteomalacic case was found in the calcium and inorganic phosphorus.

Stoichiometric-ion-product determinations on all normal and osteomalacic cases are included and indicate withdrawal of calcium from preformed bone and not a simple failure to deposit calcium.

9. The ash content of the bones of osteomalacia cases has been found to be lower than that in normal cases. The average analysis of the ash of eight normal and thirty osteomalacic cases is reported.

10. Comparative tests were conducted to determine the difference in tensile strength between normal and osteomalacic bones, it being determined that in fourteen of the former, an average of 1,669 kilos was required to produce fracture while in twenty-six of the latter, the average weight required was 1,145 kilos. The average age of all animals of both classes was approximately the same.

11. It has been noted that, while mules are much more resistant to osteomalacia than horses, the disease does occur in these animals.

12. It has been found that osteomalacia may develop within a period of approximately five months after arrival in the Philippines but may be long delayed, depending upon individual resistance and mineral constituents consumed apparently.

13. The use of the X-ray as an accurate means of diagnosis is recommended.

14. A serious outbreak of osteomalacia among Army animals in the Philippines has been controlled by the adjustment of the calcium oxide-phosphorus pentoxide intake ratio.

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TABLE 19.—*Analyses of feed.*

	Total dry matter.	Moisture.	Average digestible nutrients.			
			Protein.	Carbohydrates.	Fat.	Total.
	Per cent.	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*
Oats	[a] 90.8	[a] 9.2	[a] 9.7	[a] 52.1	[a] 3.8	[a] 70.4
Yellow native corn [b]	89.1	10.8	[c] 6.1	[c] 70.6	[c] 4.3	[c] 86.4
Palay [b]	90.6	9.4	[c] 3.9	[c] 64.0	[c] 2.2	[c] 72.9
Copra meal [b]	94.2	5.8	[c] 17.6	[c] 40.4	[c] 8.0	[c] 76.0
Wheat bran	[a] 89.9	[a] 10.1	[a] 12.5	[a] 41.6	[a] 3.0	[a] 60.9
American grain hay	[a] 88.0	[a] 12.0	[a] 4.5	[a] 38.1	[a] 1.7	[a] 46.4
Rice hay [b]	92.5	7.5	[b] 2.5	[c] 41.3	[c] 1.1	[c] 46.3
Zacate (green grass) [b]	38.5	61.5	[c] 2.7	[c] 19.2	[c] 0.8	[c] 23.7

	Average chemical composition.[b]			Ratio.	
	Ash.	CaO.	P_2O_5.	Nutritive.	Mineral.
	Per cent.	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*
Oats	3.47	0.06	0.51	1 : 6.3	1 : 8.5
Yellow native corn [b]	1.38	Trace.	0.76	1 : 13	
Palay [b]	7.01	0.11	0.59	1 : 17.5	1 : 5.4
Copra meal [b]	6.52	0.08	1.05	1 : 2.8	1 : 13.1
Wheat bran	5.17	0.07	2.40	1 : 3.9	1 : 34.3
American grain hay	5.78	0.19	0.23	1 : 9.3	1 : 1.3
Rice hay [b]	11.56	0.20	0.29	1 : 17.5	1 : 1.4
Zacate (green grass) [b]	5.05	0.12	0.23	1 : 7.7	1 : 1.9

[a] Henry and Morrison, Feeds and Feeding, 18th ed.

[b] Analysis made by Bureau of Science, Manila.

[c] Computed from analysis by Bureau of Science, Manila, and digestion coefficients of similar products published by Henry and Morrison, Feeds and Feeding, 18th ed.

TABLE 20.—*Chemical analyses of water.*

	Manila.	Corregidor.	Nichols Field.	Fort Stotsenburg.	Fort McKinley.
Color	Nil.	Nil.	Nil.	Nil.	Nil.
Odor	Nil.	Nil.	Nil.	Nil.	Nil.
Turbidity (as SiO_2)	5	Trace.	5	Trace.	5
Alkalinity (as Ca CO_3)	60	155	295	63	225
Acidity (as CO_2)	----------	25	19	3	12
Total solids	105	1,035	490	160	1,060
Appearance on ignition	----------	(a)	(a)	(a)	(a)
Silica (SiO_2)	20	120	100	90	----------
Iron and aluminum oxides ($Fe_2O_3 + Al_2O_3$)	5.6	2	3.2	2.4	4.4
Iron (Fe)	0.5	0.05	0.28	0.22	0.5
Aluminum (Al)	2.5	1	1.5	1.1	1.9
Calcium (Ca)	19.5	125	33	9.7	99
Magnesium (Mg)	5.3	46	----------	5	27
Arsenic (AS)	----------	Nil.	Nil.	Nil.	Nil.
Chlorides (Cl)	1.5	380	53	2.5	405
Bicarbonates (as H_2CO_3)	70	185	360	76	275
Sulphates (SO_4)	4	----------	10	Trace.	28
Total hardness (as $CaCO_3$)	70	505	145	45	360
Taste	----------	Normal.	Normal.	Normal.	Normal.

[a] Slight darkening.

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ILLUSTRATIONS

PLATE 1. HORSE B760

PLATE 2. X-RAY OF LEFT METACARPAL, HORSE B760

FIG. 1. November 11, 1930.
2. January 27, 1931.

PLATE 3. HORSE C442

PLATE 4. X-RAY OF LEFT METACARPAL, HORSE C442

FIG. 1. November 11, 1930.
2. January 27, 1931.

PLATE 5. HORSE 74B9

PLATE 6. X-RAY OF LEFT METACARPAL, HORSE 74B9

FIG. 1. November 11, 1930.
2. January 27, 1931.

PLATE 7. X-RAY OF LEFT METACARPAL, HORSE 76C3

FIG. 1. January 24, 1931.
2. April 24, 1931.

PLATE 8. X-RAY OF LEFT METACARPAL, HORSE O9V2

FIG. 1. January 24, 1931.
2. April, 1931.

PLATE 9. X-RAY OF LEFT METACARPAL, HORSE B776

FIG. 1. July 22, 1931.
2. October 28, 1931.

PLATE 10. MANDIBLE

PLATE 11. SKULL

PLATE 12. HEAD OF FEMUR

PLATE 13. CROSS SECTION OF VERTEBRÆ

PLATE 14. JAW

Section from the jaw showing advanced fibrous replacement, with proliferation of the fibrous tissue. There is a zone where trabeculæ still maintain their bony character just beneath the cortex. Immediately beneath this most of the trabeculæ are replaced by fibrous tissue. In the rest of the bone section there is considerable variation in the stage of the process, but all marrow spaces are replaced by this vascular new growth of fibroblastic tissue. × 15.

PLATE 15. JAW

This photograph shows an irregular distribution of the most advanced stages of the process, but all the bone lamellæ are modified in greater or less degree, some of them being completely replaced by fibrous tissue. The least-marked change appears to be just beneath the cortex. × 15.

PLATE 16. MAXILLA

Photograph taken near the base of the larger section. There are many osteoclastic giant cells. This photograph shows the terminal stages in the absorption of the bony lamellæ. × 300.

PLATE 17. MAXILLA

The section includes the pericortical fibrous tissue. There is some erosion of the lamellæ, but bone cells are still distinct. The lamellar spaces are completely filled with vascular fibrous tissue. × 300.

PLATE 18. HEAD OF FEMUR

Low-power photograph showing most of the section from the head of the femur. Advanced bony replacement by fibrous tissue in the center of the picture. Spaces contain marrow at the right and left. Necrotic bone is seen apparently in the marrow spaces. This appearance is caused by the section passing through partially necrosed lamellæ parallel to their surface, which were at right angles to the other lamellæ, which apparently form the boundaries of the spaces. These are particularly well shown to the left of the picture. × 15.

PLATE 19. HEAD OF FEMUR

Section shows practically normal marrow at the top and a partially necrosed bone trabecula, center, and at the bottom the section has passed through another trabecula parallel to its surface, this surface being at right angles to the trabecula in the center. There is some marrow in this space also. × 300.

PLATE 20. HEAD OF FEMUR

Photograph taken from section where some marrow still exists. Slightly degenerated bone lamellæ, showing absorption of the lamellar cortex, with a few osteoclasts, and a deposit of fibrous tissue along the bone about the periphery of the marrow space. The marrow appears essentially normal. × 300.

PLATE 21. HEAD OF FEMUR

Photograph from an area of advanced replacement fibrosis, with many osteoclastic giant cells. × 300.

PLATE 22. BONE AND MARROW, LARGE METACARPAL, MIDDLE THIRD

Photograph of the central portion of the section showing loss of lime salts in the bone adjacent to the marrow cavity. This is indicated by the paler staining. The cortical tissue and the marrow have been lost through technical procedure.

PLATE 23. X-RAY FINDINGS

FIG. 1. Normal.
2. Slight rarefaction.

PLATE 24. X-RAY FINDINGS

FIG. 1. Moderate rarefaction.
2. Severe rarefaction.

TEXT FIGURES

FIG. 1. Admission ratios: "Diseases of Bones and of the Organs of Locomotion."
2. Admission rates for conditions indicated in Table 3.
3. Comparison of osteomalacia admission rates with total admission rates.
4. Comparison of admissions for osteomalacia and admissions for all other causes
5. Number of cases and percentage of total in age groups of two years each.
6. Leucocyte count and temperature chart of horse 1C09.
7. Feeding experiment 1.
8. Feeding experiment 2.
9. Feeding experiment 3.
10. Feeding experiment 4.
11. Comparison of American and Philippine rations.
12. Ash content of normal and osteomalacic bones.
13. Disposition of osteomalacia cases at Fort Stotsenburg from July 1, 1930, to July 1, 1931 (340 cases).
14. Age groups of animals destroyed for osteomalacia at Fort Stotsenburg.
15. Comparison of osteomalacia incidence and calcium in water supply.
16. Total admission rates and rates for osteomalacia at Fort Stotsenburg.

PLATE 1. HORSE B760.

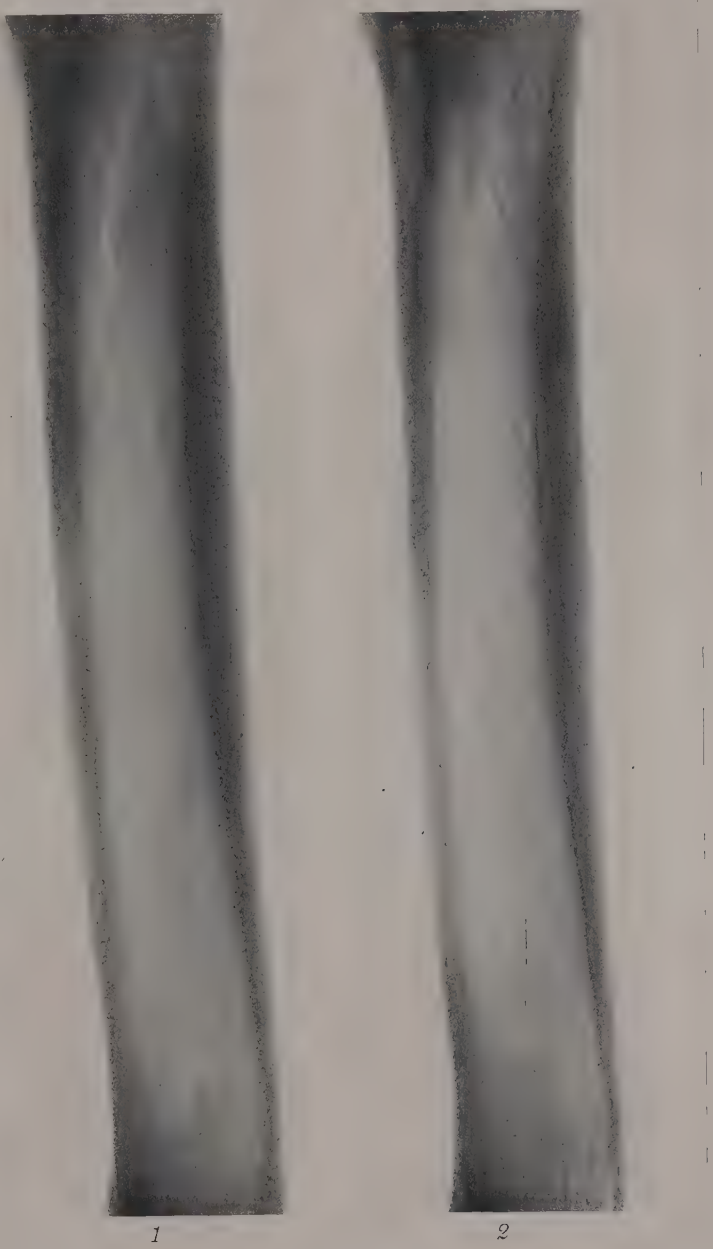

PLATE 2. X-RAY OF LEFT METACARPAL, HORSE B760. 1, NOVEMBER 11, 1930; 2, JANUARY 27, 1931.

PLATE 3. HORSE C442.

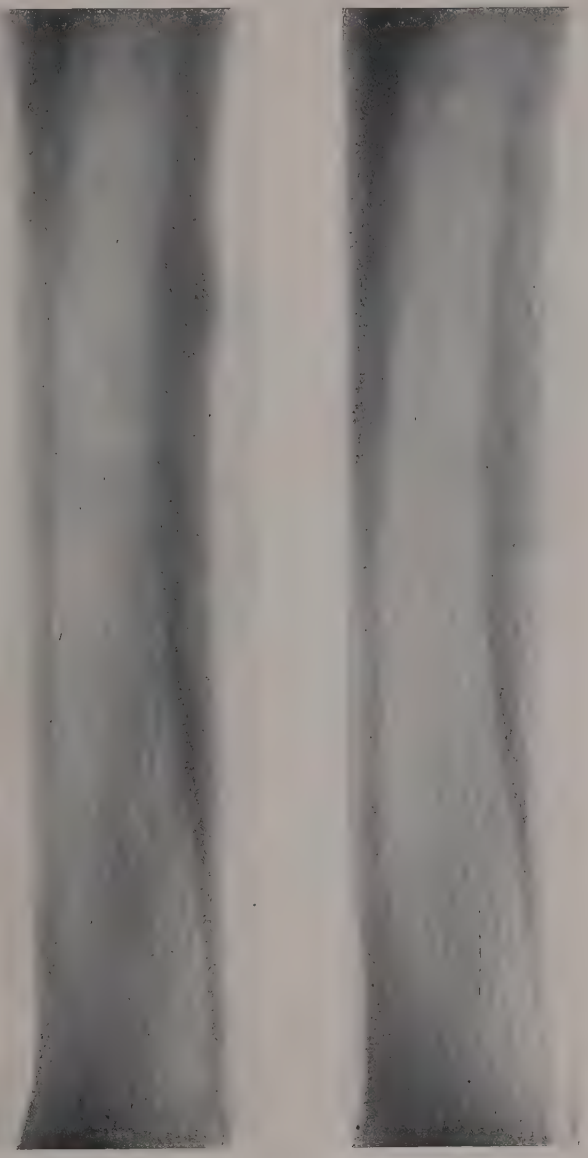

PLATE 4. X-RAY OF LEFT METACARPAL, HORSE C442. 1, NOVEMBER 11, 1930; 2, JANUARY 27, 1931.

PLATE 5. HORSE 74B9.

PLATE 6. X-RAY OF LEFT METACARPAL, HORSE 74B9. 1, NOVEMBER 11, 1930; 2, JANUARY 27, 1931.

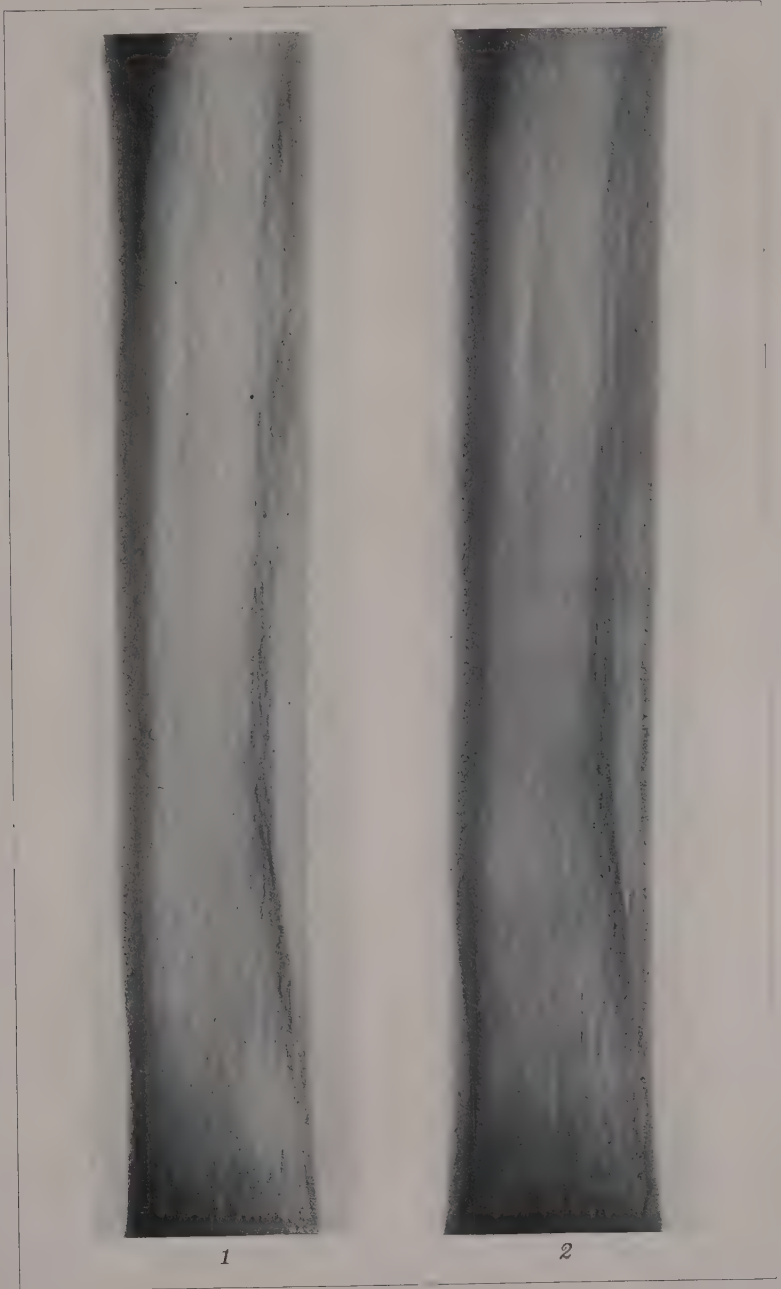

PLATE 7. X-RAY OF LEFT METACARPAL, HORSE 76C3. 1, JANUARY 24, 1931; 2, APRIL 24, 1931.

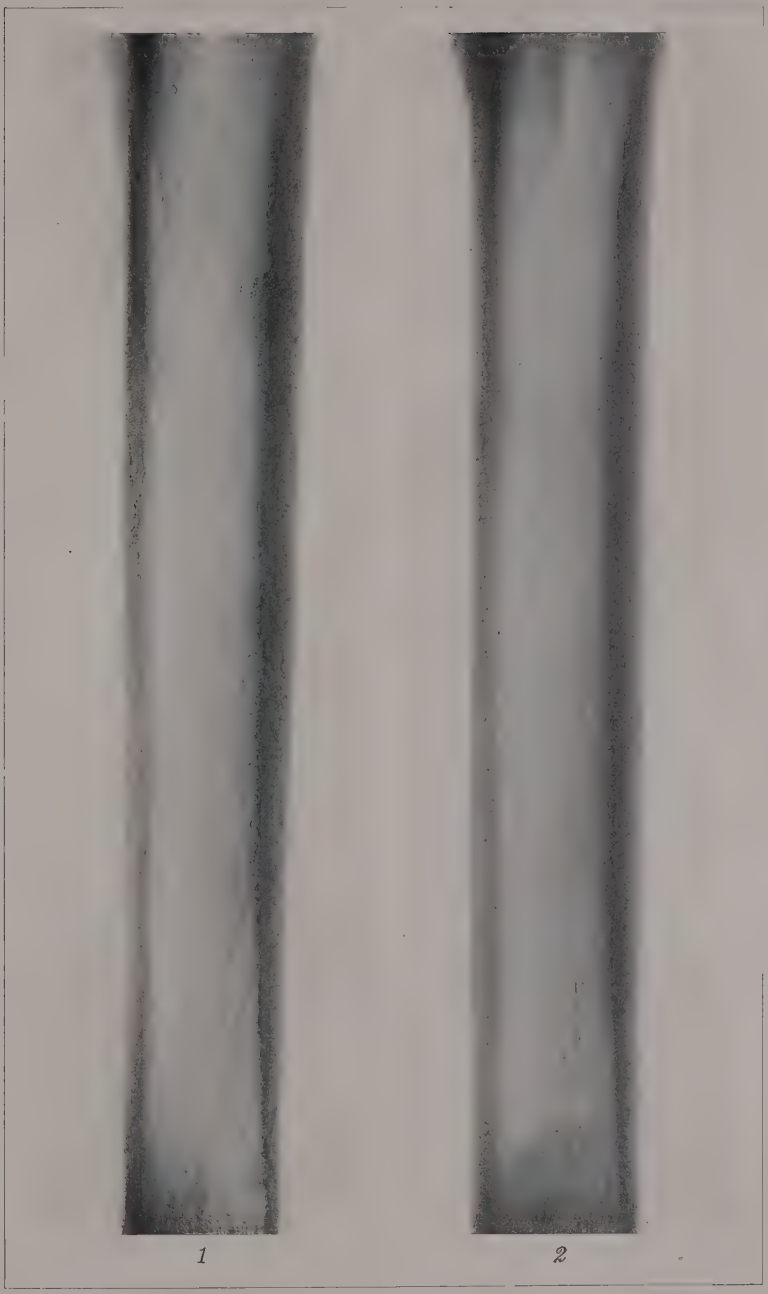

PLATE 8. X-RAY OF LEFT METACARPAL, HORSE 09V2. 1, JANUARY 24, 1931; 2, APRIL 24, 1931.

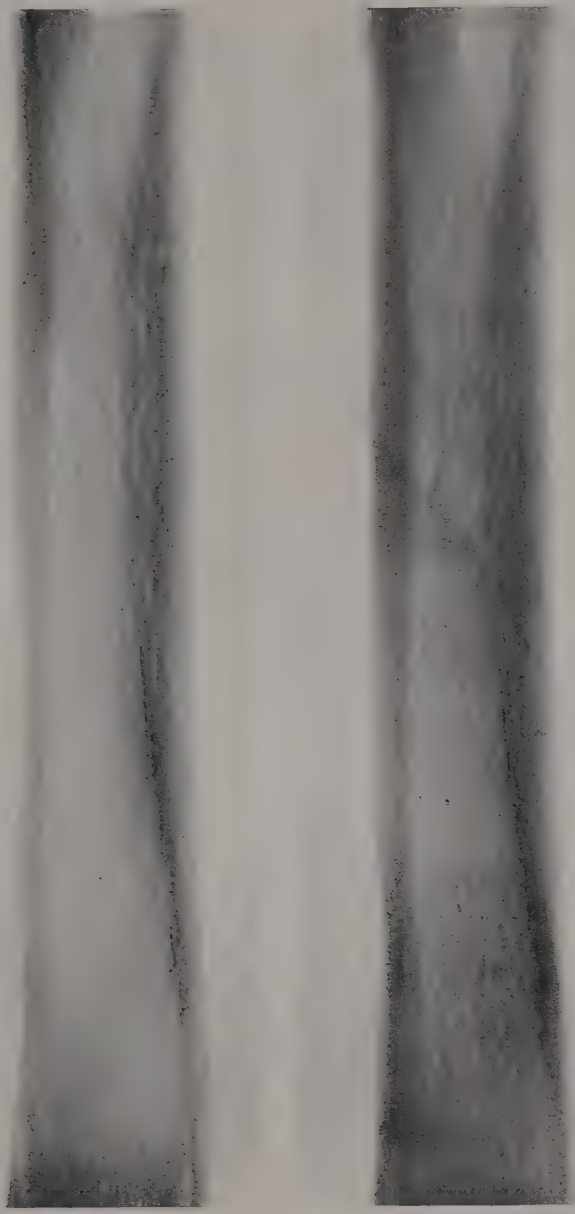

PLATE 9. X-RAY OF LEFT METACARPAL, HORSE B776. 1, JULY 22, 1931; 2, OCTOBER 28, 1931.

PLATE 10. MANDIBLE.

PLATE 11. SKULL.

PLATE 12. HEAD OF FEMUR.

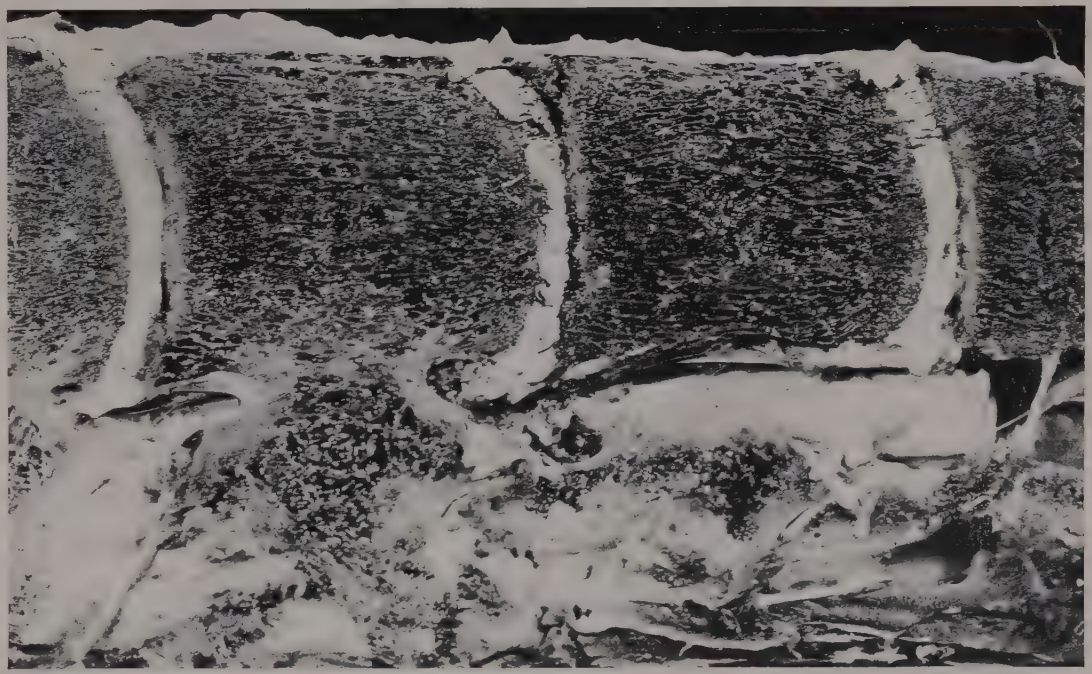

PLATE 13. CROSS SECTION OF VERTEBRAE

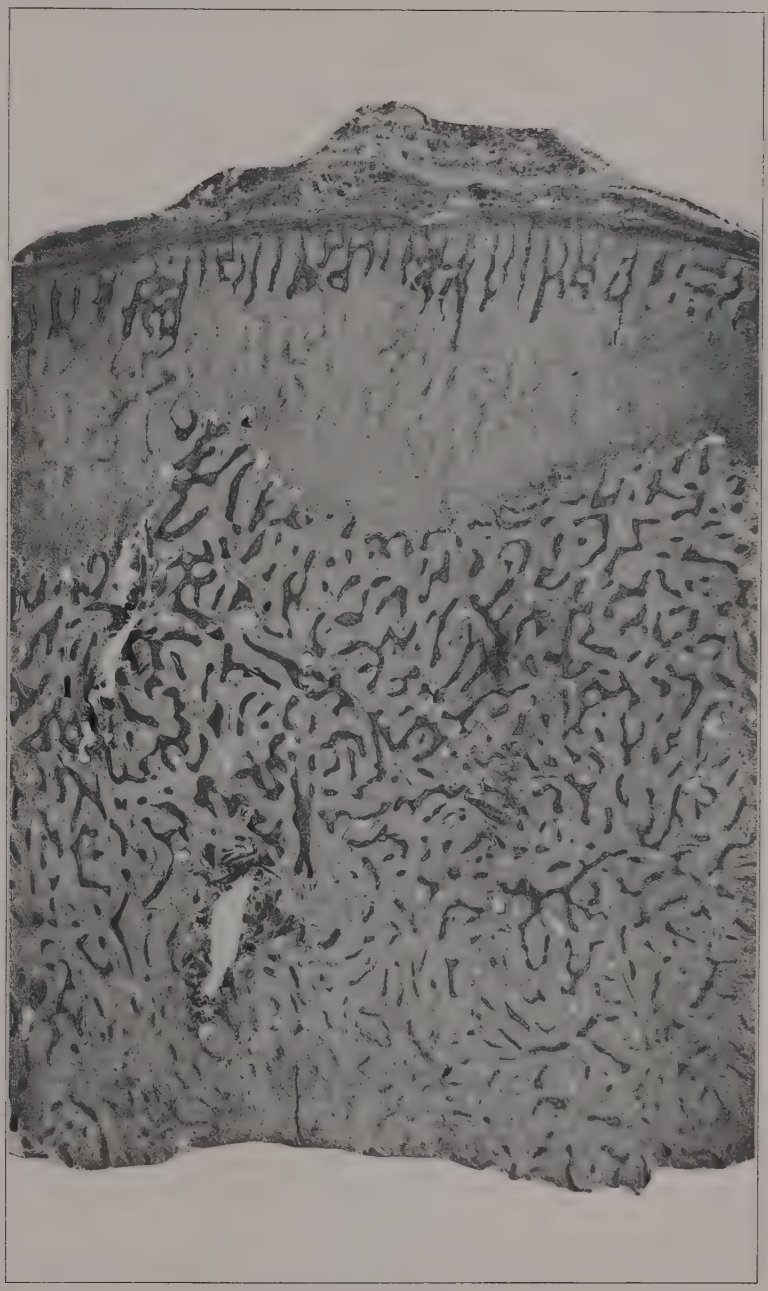

PLATE 14. JAW.

PLATE 15. JAW.

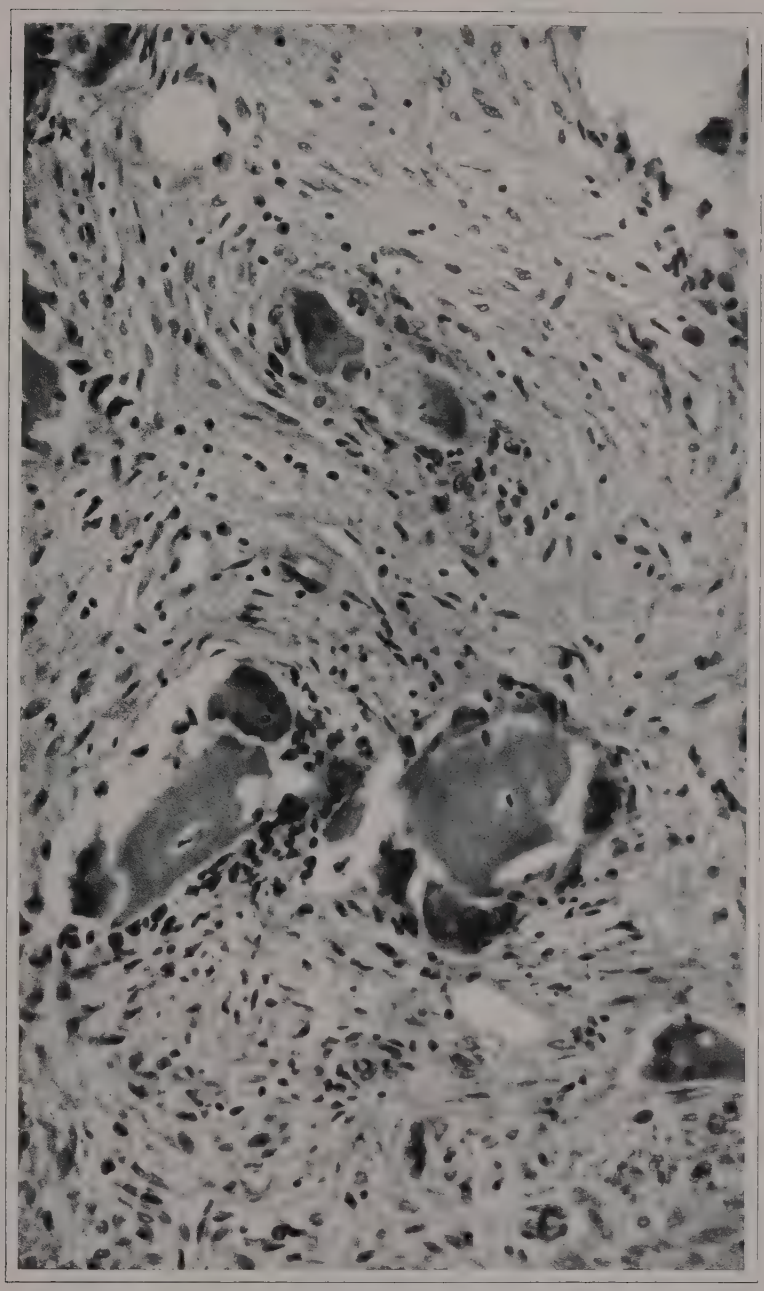

PLATE 16. MAXILLA.

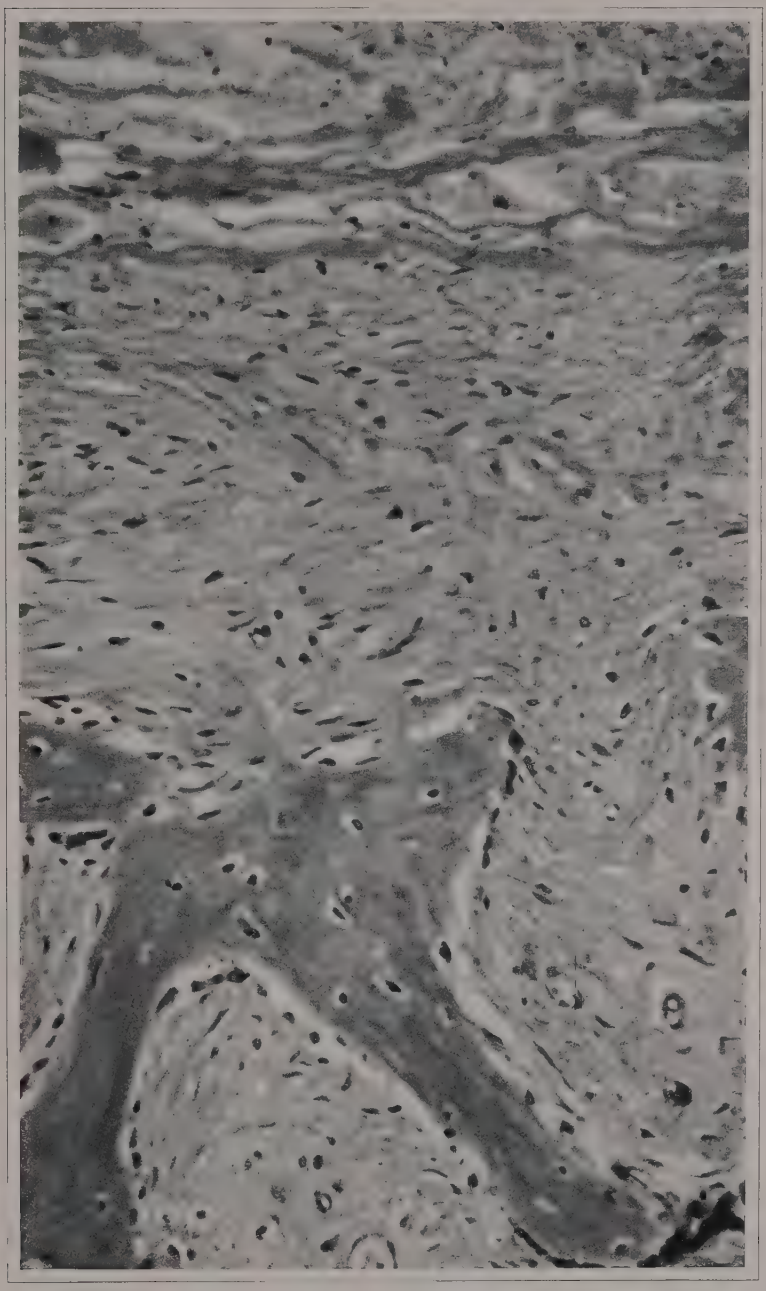

PLATE 17. MAXILLA.

PLATE 18. HEAD OF FEMUR.

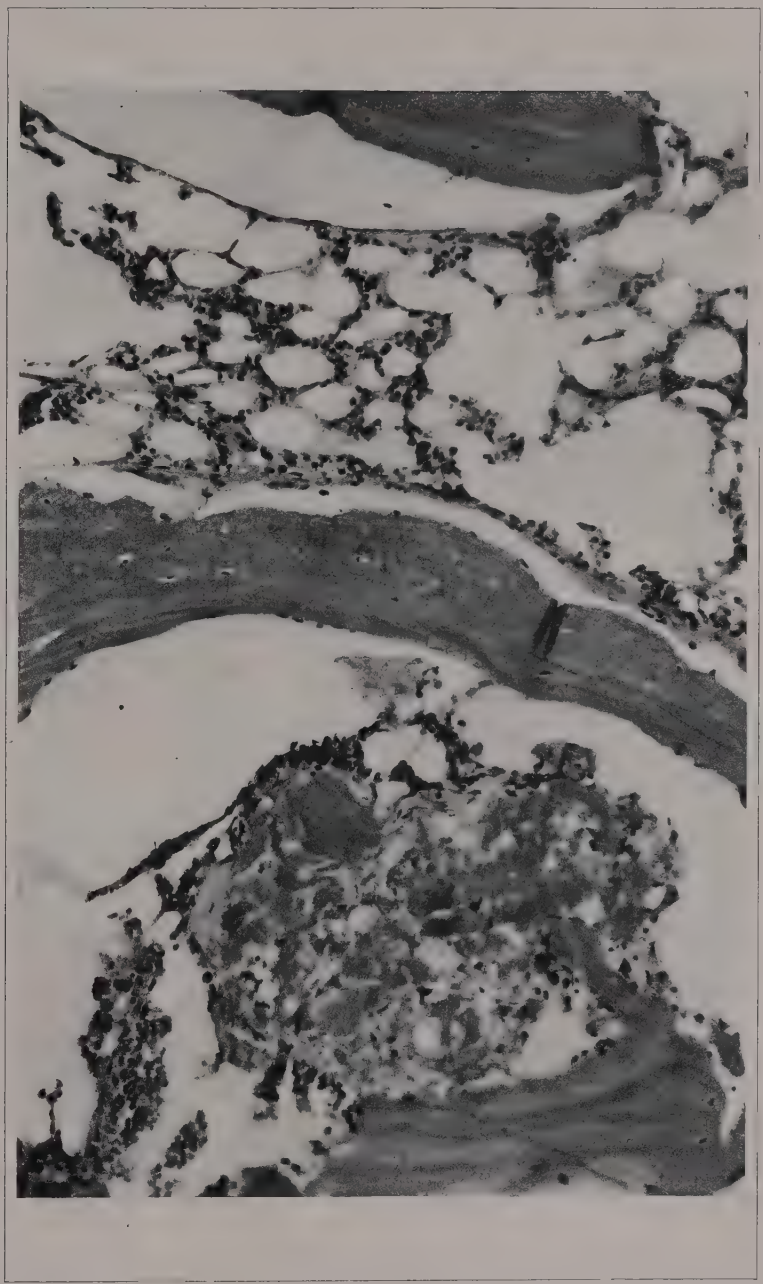

PLATE 19. HEAD OF FEMUR.

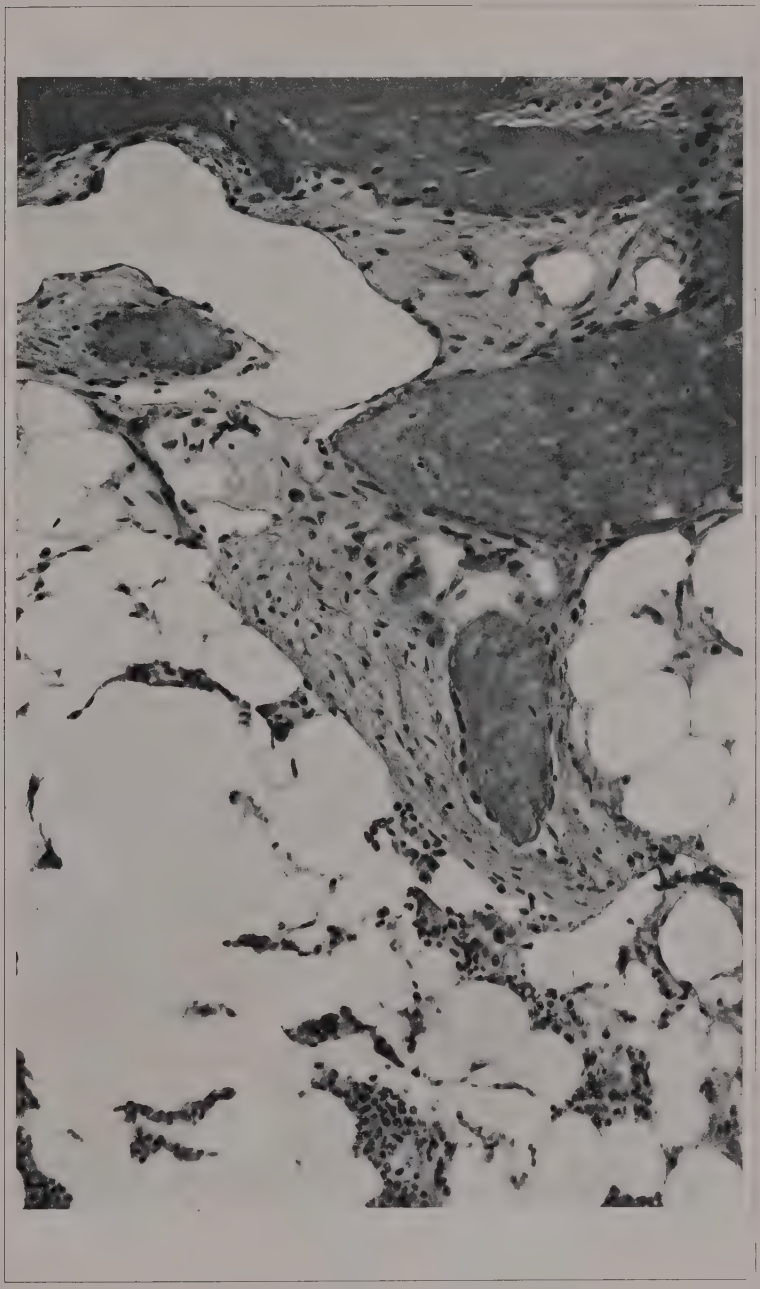

PLATE 20. HEAD OF FEMUR.

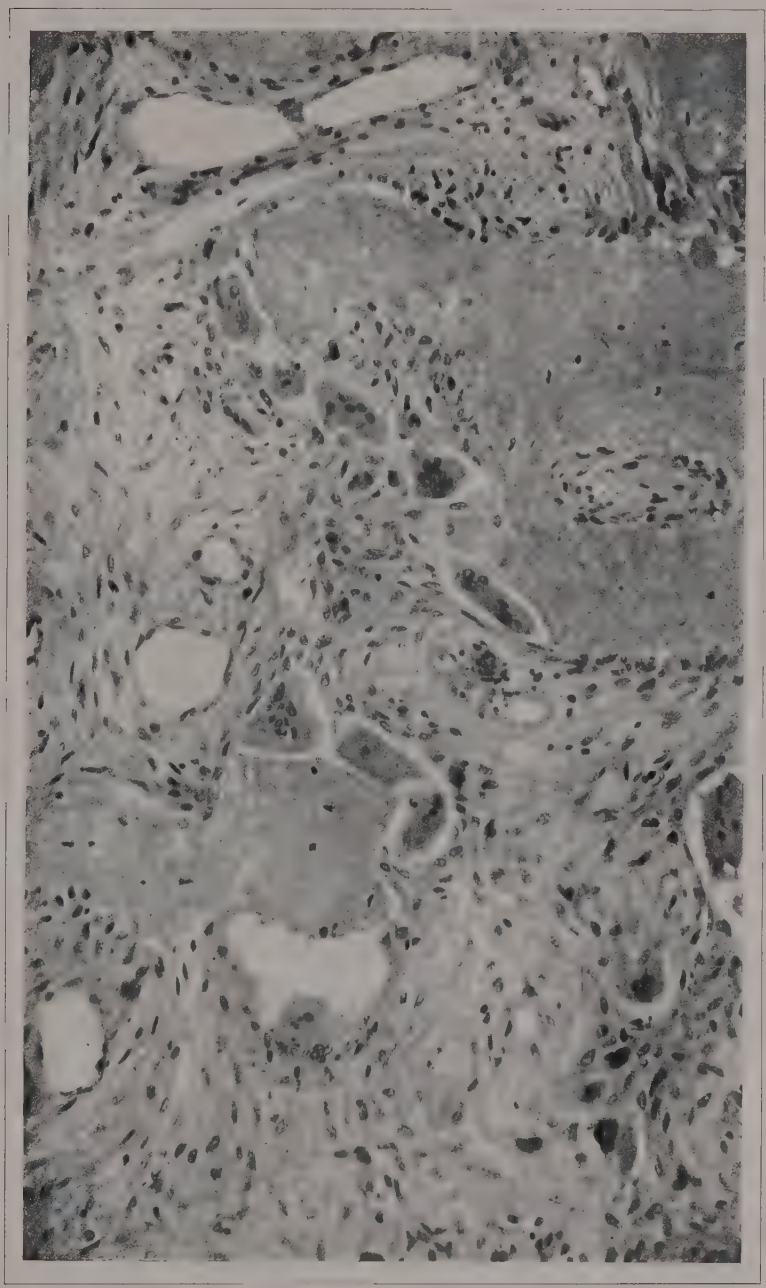

PLATE 21. HEAD OF FEMUR.

PLATE 22. BONE AND MARROW, LARGE METACARPAL, MIDDLE THIRD.

PLATE 23. X-RAY FINDINGS. 1, NORMAL; 2, SLIGHT RAREFACTION.

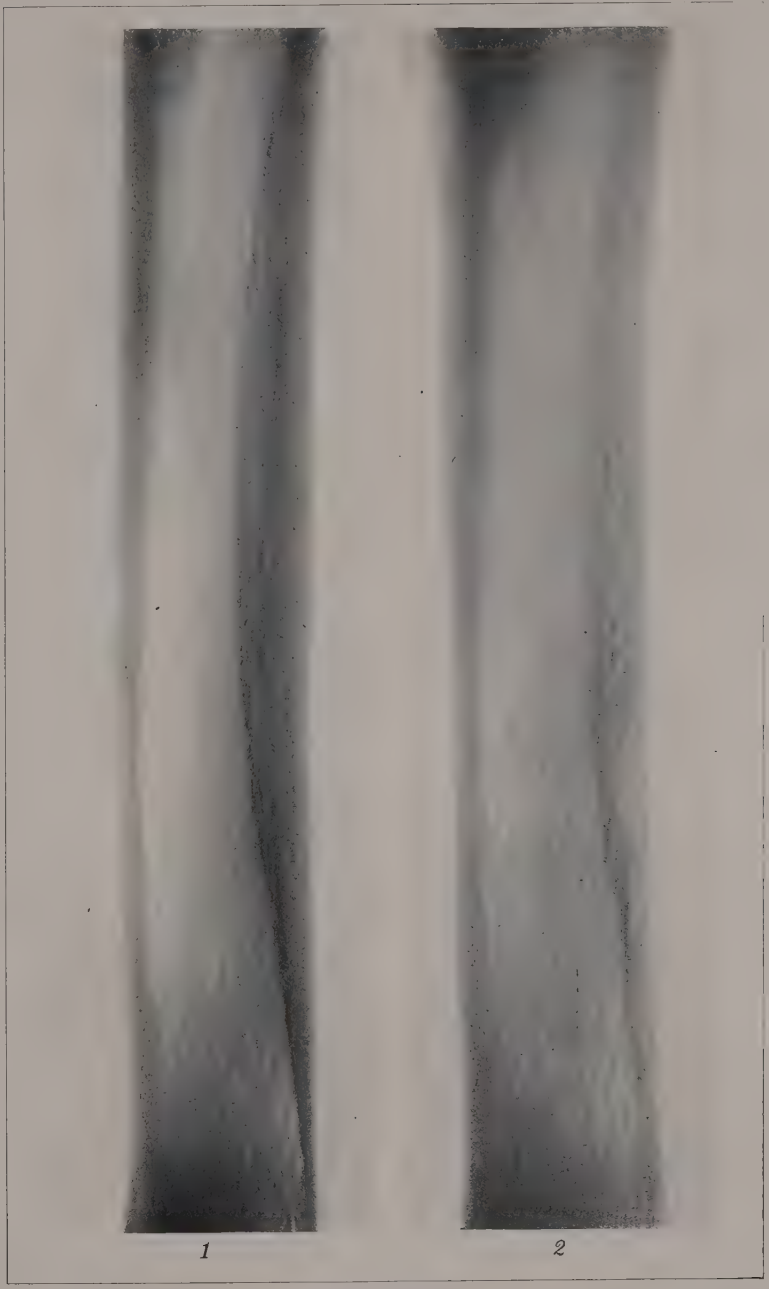

PLATE 24. X-RAY FINDINGS. 1, MODERATE RAREFACTION; 2, SEVERE RAREFACTION.

AMBASSIDÆ FROM THE PHILIPPINES

By L. F. DE BEAUFORT

Of Amsterdam, Holland

Through the kindness of Dr. William H. Brown, director of the Bureau of Science, Manila, I have had the opportunity to study a collection of more than one thousand specimens of the genus *Ambassis*.

The collection contains no species previously unknown from the Philippines, excepting *Ambassis miops* Günther. Although there is nothing new in this respect, I have been able, thanks to the large amount of material, to get a better notion of the value of various species. This will be discussed under each species.

In the matter of synonymy I have quoted only the first author and Weber and de Beaufort;[1] full synonymy can be found in the latter work. When it was necessary for the discussion, I have cited more fully.

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Ambassis urotaenia DE BEAUFORT, Bijdragen Dierk. afl. 19 (1913) 112.

Ambassis urotaenia WEBER and DE BEAUFORT, 404.

Localities.—Vigan, Ilocos Sur Province; Buyon River, Polillo; Lake Taal and Batangas, Batangas Province; Puerto Galera and Pinamalayan, Mindoro; Arimbay River and Bigaa, Albay Province; Bacon and Bulan, Sorsogon Province; Despujols, Tablas; Bantayan, Cebu Province; Loay, Bohol; Lazi, Siquijor; Culion and Balabac Islands, Palawan Province; Placer, Agusan River, Cagayan, Zamboanga, Caldera Bay, and Davao, Mindanao.

As I have already explained[2] *Ambassis lungi* is a synonym of this species. The large amount of material from the Philippines has convinced me further that my former opinion was right.

[1] Fishes of the Indo-Australian Archipelago 5 (1929).

[2] Loc. cit.

Ambassis urotaenia and the following species are easily distinguished from the others by having only 8 to 9 scales between nape and dorsal. It is characterized by having only one row of scales on the cheeks. It has a wide distribution in Indic and Western Pacific waters.

2. AMBASSIS KOPSI Bleeker.

Ambassis kopsii BLEEKER, Nat. Tijdschr. Ned. Indië 15 (1858) 253.
Ambassis kopsi WEBER and DE BEAUFORT, 405.

Localities.—Paombong, Bulacan Province; Orani, Bataan Province; Malabon, Rizal Province; Manila; Cavite, Cavite Province; Mangarin, Mindoro; Catbalogan, Samar; Estancia and Dumangas, Iloilo Province; Polo Plantation, Oriental Negros Province; Guinlo and Balabac, Palawan Province; Buan Island, Sulu Archipelago. There were also specimens from Sandakan, Borneo, in the collection.

This species, which is easily distinguished from *A. urotaenia* by the greater depth of the head, by having two rows of scales on the cheeks, and by the black tip of the spinous dorsal, has a very small habitat in comparison with the former, as it is only known from Singapore, Banks, Borneo, and the Philippines, including Palawan and Sulu Archipelago.

3. AMBASSIS COMMERSONI Cuvier and Valenciennes.

Ambassis commersonii CUVIER and VALENCIENNES, Hist. Nat. Poissons 2 (1828) 176.
Ambassis commersoni WEBER and DE BEAUFORT, 406.

Localities.—Buguey, Cagayan Province; Vigan, Ilocos Sur Province; Iba, Zambales Province; Lake Taal, Batangas Province; Camarines Sur Province; Baco River, Mindoro; San Jose de Buenavista, Antique Province; Agusan River, Zamboanga, Mindanao River, Cotabato, and Davao, Mindanao.

Ambassis commersoni is very close to *A. miops*, and after having examined the extensive material from the Philippines, I am not sure that they are distinct. Both belong to a group of species in which the linea lateralis is constantly continuous. They differ chiefly in the number of scales between nape and dorsal, which is always greater than in the two preceding species and amounts to 17 to 22 in *A. commersoni* and 12 to 16 in *A. miops*. Furthermore, the maxillary is somewhat dilated distally and emarginated posteriorly in *A. commersoni*, whereas the maxillary of *A. miops* is obliquely truncated behind and scarcely dilated. In this way it is rather easy to distinguish between typical specimens of *A. commersoni* and of *A. miops*,

but we found also specimens with 16 or 17 scales before the dorsal and with a maxillary that stands in shape between those of the two species. For discussion see the next following species.

4. AMBASSIS MIOPS Günther.

Ambassis miops GÜNTHER, Proc. Zool. Soc. London (1871) 655.
Ambassis miops WEBER and DE BEAUFORT, 408.

Localities.—Bauang River, La Union Province; Buyon River, Polillo; Cavite, Cavite Province; Lake Taal and Pansipit River, Batangas Province; Baco, Lapog, Bato, and Naujan Rivers, and Butas, Mindoro; Arimbay River and Bigaa, Albay Province; Bacon, Sorsogon Province; Sibuyan, Romblon Province; Dumarao, Capiz Province; Dumaguete and Zamboanguita, Oriental Negros Province; Anajauan, Leyte; Malampaya Sound and Balabac, Palawan Province; Cagayan, Zamboanga, Mindanao River, and Davao, Mindanao.

As said above, some specimens are intermediate between this species and *A. commersoni*. *Ambassis commersoni* is distributed from Madagascar and the east coast of Africa to New Guinea and North Australia, *A. miops* from Madras to Rara Tonga. It may be that they consist of the western and eastern subspecies of the same species, but it may just as well be that the two have not always been distinguished and that both occur in the whole area. It is possible that Bleeker has mixed these two species, for in his description of *A. commersoni*[3] he mentions about 13 scales before the dorsal (which would point to *A. miops*), whereas we found 18 to 20 prædorsal scales in the specimens of Bleeker's collections in the Leiden Museum, labelled by him *A. commersoni*. *Ambassis miops* is not mentioned by Bleeker in the Atlas. If further investigations show that really *A. miops* is absent from the western part of the Indic, and that *A. commersoni* does not reach so far into the Pacific as *A. miops*, I think the only explanation possible is that they hybridize where they occur together. We will meet with more species of *Ambassis* that seem to merge into each other.

A third species of this group, *A. nalua*, has been recorded from the Philippines by Bleeker, but I did not find it in the material sent to me. It may be distinguished from *A. miops* and *A. commersoni* by having the maxillary much more dilated posteriorly than *A. commersoni* and by having only 12 or 13 prædorsal scales.

[3] Atlas Ichthyologique 8: 136.

5. AMBASSIS GYMNOCEPHALUS (Lacépède).

Lutjanus gymnocephalus LACÉPÈDE, Hist. Nat. Poissons **3** (1802) pl. 23. fig. 3; **4** (1802) 216.

Ambassis gymnocephalus WEBER and DE BEAUFORT, 412.

Localities.—Malabon, Rizal Province; Tondo Market, Manila; Lake Taal, Batangas Province; San Miguel Bay, Camarines Sur Province; Catbalogan, Samar; Carigara, Leyte; Bantayan, Cebu Province; Guimaras Strait. There are further specimens in the collection from Sandakan, Borneo, and from Amoy and Hong Kong.

Ambassis gymnocephalus is easily distinguished by its interrupted lateral line and having more than one spine on the supraorbital ridge. This ridge is smooth excepting a posterior terminal spine in all species of *Ambassis* known to me, but in *gymnocephalus* it is preceded by one, two, or three spines. As Weber and de Beaufort have remarked before, *A. gymnocephalus* is closely related to *A. buruensis*. In the material from the Philippines I found several specimens in which one of the supraorbital ridges had only a terminal spine, whereas the other had two spines. As there are no other salient differences between *A. gymnocephalus* and *A. buruensis* (the difference in depth is not constant), I am at a loss how to decide whether these specimens belong to *gymnocephalus* or to *buruensis*. Here, again, the possibility of interbreeding may be thought of.

6. AMBASSIS INTERRUPTA Bleeker.

Ambassis interrupta BLEEKER, Nat. Tijdschr. Ned. Indië **3** (1852) 696.

Priopis buruensis JORDAN and SEALE, Bull. Bur. Fish. **26** (1906) (1907) 18.

Ambassis interrupta WEBER and DE BEAUFORT, 415.

Localities.—Agno River, Pangasinan Province; Subic, Zambales Province; Guagua and Pilipit Creek, Pampanga Province; Orani and Pilar, Bataan Province; Buyon River, Polillo; Malabon, Rizal Province; Pasig River, Bureau of Science fishpond, and Manila markets, Manila; Cavite, Cavite Province; Balanak River, Laguna Province; Laguna de Bay; Pansipit River, Batangas Province; Arimbay River and Bigaa, Albay Province; Bato River, Mindoro; Anajauan, Leyte; Agusan River, Cagayan, Zamboanga, and Davao, Mindanao; Balabac, Palawan Province; Malum River and Tawitawi, Sulu Archipelago; and further specimens from Hong Kong.

As mentioned by Jordan and Richardson,[4] *buruensis* and *interrupta* are hardly distinguishable. The difference between these two species is, that in *A. interrupta* the border of the interoperculum is toothed, whereas it is smooth or with a single spine in *buruensis*. Here, too, we find all kinds of gradations between specimens with well-developed spines and others with these spines almost obsolete. This is no matter of age, as some very small specimens have strong teeth and others, equally small, have none.

There is not much difference between the range of these two species. *Ambassis interrupta* covers a somewhat larger area, being known from the Andamans, the Indo-Australian Archipelago, the Philippines, and New Caledonia. *Ambassis buruensis* is only known from the Indo-Australian Archipelago and the Philippines. This shows the improbability of the differences between the two being due to sex.

7. AMBASSIS BURUENSIS Bleeker.

Ambassis buruensis BLEEKER, Nat. Tijdschr. Ned. Indië 11 (1856) 396.

Ambassis buruensis WEBER and DE BEAUFORT, 417.

Localities.—Bauang River, La Union Province; Subic, Zambales Province; Pasig River, Manila; Lake Taal, Pansipit River, and Balayan Bay, Batangas Province; Baco and Naujan Rivers, Mindoro; Arimbay River and Bigaa, Albay Province; San Jose de Buenavista, Antique Province; Cabalian, Leyte; Dumaguete, Oriental Negros Province; Lazi, Siquijor; Cagayan, Agusan River, and Caldera Bay, Mindanao.

We have seen that *A. buruensis, A. interrupta,* and *A. gymnocephalus* are closely allied species. They have in common the interrupted linea lateralis. *Ambassis buruensis* is the less "spiny" of the three, having neither spines on the supraorbital ridge nor on the lower border of the interoperculum, but we have seen that in some specimens two spines may occur on the supraorbital ridge, and that in others traces of spines on the interoperculiar border may be found. In one direction *A. buruensis* seems to vary towards *A. gymnocephalus,* in the other towards *A. interrupta.* In this respect it is interesting to remember that Weber and de Beaufort[5] record a specimen of

[4] Bull. U. S. Bur. Fish. 27 (1907) (1908) 255.

[5] Op. cit. 413, footnote.

A. gymnocephalus of unknown locality, with a serrated interoperculum. Thus, we have seen specimens combining the characters of *buruensis* and *interrupta*, of *buruensis* and *gymnocephalus*, and of *gymnocephalus* and *interrupta*.

Of the three species *A. gymnocephalus*, *A. buruensis*, and *A. interrupta*, the first has by far the largest distribution, ranging from the coast of East Africa to the north coast of Australia. We have seen that the range of *interrupta* and *buruensis* is almost restricted to the Indo-Australian Archipelago and its neighborhood. Might it be possible that the occurrence here in coastal and brackish waters of isolated islands has promoted the origin of different strains?

SOME STUDIES IN THE LARVICIDAL EFFECTS OF ARSENICALS OTHER THAN PARIS GREEN AGAINST ANOPHELES LARVÆ LARVICIDE STUDIES, III [1]

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The following studies are a continuation of those reported in our first two papers.[2] These experiments are being made in an attempt to reduce the cost of malaria control as regards larvicidal measures, possibly by finding a cheaper poison than Paris green, perhaps by making it possible to lessen the amount necessary to be effective, or by devising more-efficient measures of application.

In the first paper the use of powdered charcoal as a diluent for Paris green was considered. In the second paper laboratory experiments were reported in which Paris green partially adsorbed on charcoal in various concentrations and combinations, was tried. In this paper arsenicals other than Paris green are considered. The results obtained indicate that, as a mosquito larvicide, Paris green is more efficient than the other arsenical preparations we used. This suggests that the copper in Paris green has a real influence in the excellent larvicidal effects of this substance.

EXPERIMENTAL PROCEDURE

The method of preparing the charcoal larvicides and the technic employed in using them have been discussed in our pre-

[1] These studies were undertaken by the divisions of organic chemistry and of malaria investigations, Bureau of Science. The division of malaria investigations is coöperatively supported by the Bureau of Science and by the International Health Division of the Rockefeller Foundation.

[2] Russell, P. F., and A. P. West, Philip. Journ. Sci. 48 (1932).
West, A. P., and P. F. Russell, Philip. Journ. Sci. 48 (1932).

vious papers. It is obviously important that standardized methods be used if valid comparisons are to be made. Consequently care has been taken to follow a given procedure in all experiments.

Only third- and fourth-stage anopheles larvæ were used, chiefly of the species *Anopheles subpictus* and *A. hyrcanus* var. *sinensis*. Artesian water was placed in enamel pans so that the surface area was approximately 532 square centimeters and the depth 5 centimeters. Larvæ were not removed as dead until they failed to respond to stimulation, all motion having ceased. They were then transferred to beakers of water for observation to insure against mistakes in diagnosis of death-point or in counting. Comparable amounts of larvicide were used, and the weight in grams is given in the tables.

RESULTS

In Table 1 are presented the results of some tests of the larvicidal properties of arsenic trioxide adsorbed on charcoal. These experiments were made to find out if this simple arsenical in combination with charcoal will poison larvæ as effectively as Paris green. The latter contains copper as well as arsenic and is consequently more expensive.

These tests, when compared with those of Paris green (see our second paper, Tables 1 to 5) show plainly that Paris green is more effective than arsenic trioxide. Not until the concentration of arsenic trioxide reached 8.5 per cent (tests 2, 51, and 68) did the larvicide kill 100 per cent of the larvæ in twenty-four hours. Even concentrations as high as 10 per cent (tests 3, 13, and 52) failed to eliminate the larvæ completely.

Incomplete effect was also seen when the arsenic trioxide was mechanically mixed with charcoal as a diluent in a 1 per cent concentration (test 85). These results are in line with those of Barber and Hayne.[3] In Table 2 are presented the results of tests in which lime, borax, or sodium carbonate, in addition to arsenic trioxide, were adsorbed on charcoal. Here again the results are notably less satisfactory than when Paris green was used in similar combinations as shown in our second paper.

This is suggestive evidence that the copper in the Paris green has a real influence in the larvicidal effects of this substance. Such a suggestion is not original but has been broached by

[3] U. S. Pub. Health Rep. 36, No. 49 (1921) 3028.

others. For example, Williams in discussing a paper by Ginsburg [4] refers to this possibility.

In Table 3 are shown the results of tests with miscellaneous arsenical preparations adsorbed on charcoal in various combinations and concentrations. Only zinc arsenite (test 156) gave results comparable to Paris green.

It is interesting that atoxyl (test 106), arsenic pentoxide (test 88), and salvarsan (tests 104 and 105) were relatively harmless.

It is surprising that such highly toxic substances as barium arsenite (test 31) and lead arsenite (test 62) were comparatively harmless to anopheles larvæ in concentrations equal to or greater than those which in the case of Paris green were lethal for the larvæ. This, however, simply confirms the results of Barber and Hayne. Tannic acid in combination with arsenic trioxide (test 58) was ineffective.

In all of these experiments the larvæ had no difficulty in filling their digestive tract with the charcoal, and after one to two hours the tract was continuously filled with this material for twenty-four hours; in some cases, for seventy-two hours, as in tests 31 and 58.

SUMMARY

Some experiments are reported dealing with the larvicidal effects of certain arsenicals, other than Paris green, when applied to charcoal. These studies confirm the belief that Paris green is the arsenical larvicide par excellence.

The fact that Paris green is more efficient, as a mosquito larvicide, than other arsenical preparations suggests that the copper in Paris green has a real influence in the excellent larvicidal effects of this substance.

[4] Proc. 15th Ann. Meeting New Jersey Mosquito Extermination Assoc., New Brunswick, N. J. (1928) 63.

TABLE 1.—*Larvicidal effects of arsenic trioxide partially adsorbed on charcoal.*

Test No.	Substances used.	Concentration.	Number of larvæ used.	Percentage of dead larvæ in time periods. Cumulative totals.																Weight of larvicide.
				Minutes.								Hours.								
				15	30	45	60	75	90	105	120	2.5	3	4	5	6	7	24	48	72	
		Per cent.																		*g.*
57	Nothing (control)		50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
53	Arsenic trioxide	0.55	50	0	0	0	0	2	2	2	2	2	2	2	2	2		16			0.13
1	do	7.0	50	0	0	0	0	0	2	2	4	12	18	46	58	72		88			0.13
2	do	8.5	50	0	0	2	2	6	8	16	28	38	60	78	80	82		92			0.13
51	do	8.5	50	0	0	2	4	8	26	42	54	64	74	92	96	96		100			0.13
68	do	8.5	51	0	0	0	0	2	2	4	8	10	20	24	28	36		88			0.13
3	do	10.0	50	0	0	2	2	6	6	20	32	44	64	84	86	92		98			0.13
13	do	10.0	50	0	0	2	2	2	2	2	8	12	18	44	56	64		90			0.01
52	do	10.0	50	0	0	0	2	2	8	14	20	22	26	38	56	62		94			0.13
[a] 83	{Arsenic trioxide / Charcoal 1/1000} mixture	0.1	50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0			0.1
[a] 84	{Arsenic trioxide / Charcoal 1/200} mixture	0.5	50	0	0	0	0	0	0	2	2	4	4	6	14	16	22	24			0.1
[a] 85	{Arsenic trioxide / Charcoal 1/100} mixture	1.0	50	0	0	0	0	0	2	4	6	14	16	16	20	24	34	72			0.1

[a] In tests 83, 84, and 85 the arsenic trioxide powder was mixed with the charcoal and not adsorbed on it.

TABLE 2.—*Larvicidal effects of arsenic trioxide and lime, borax, or sodium carbonate partially adsorbed on charcoal.*

Test No.	Substances used.	Concentration.	Number of larvæ used.	Percentage of dead larvæ in time periods. Cumulative totals.																Weight of larvicide.
				Minutes.								Hours.								
				15	30	45	60	75	90	105	120	2.5	3	4	5	6	7	24	48	72	
		Per cent.																		*g.*
38	Nothing (control)		50	0	0	0	0	0	0	0	0	0	0	0	0	0	----	0	0	0	
66	Arsenic trioxide	4.0	50	0	0	0	0	0	2	2	2	6	12	16	40	46	46	54	----	----	0.1281
	Lime	1.0																			
74	Arsenic trioxide	4.0	49	0	0	0	0	0	2	10	16	16	30	32	42	56	----	76	----	----	0.1281
	Lime	1.0																			
46	Arsenic trioxide	8.0	50	2	4	4	8	16	22	28	40	44	52	60	74	80	----	98	----	----	0.1281
	Lime	1.0																			
47	Arsenic trioxide	10.0	50	0	2	2	4	4	14	22	28	30	34	40	52	58	----	94	----	----	0.1281
	Lime	1.0																			
67	Arsenic trioxide	4.0	50	0	0	0	0	0	0	0	2	2	2	2	4	6	6	6	----	----	0.1281
	Sodium carbonate	1.0																			
75	Arsenic trioxide	4.0	47	0	0	0	0	0	0	2	2	2	6	16	24	30	----	45	----	----	0.1281
	Sodium carbonate	1.0																			
101	Arsenic trioxide	0.8	50	0	0	0	0	0	0	0	0	0	0	0	0	0	6	8	----	----	0.1
	Borax	0.4																			
76	Arsenic trioxide	1.0	50	0	0	0	0	0	0	0	0	0	0	0	0	----	----	4	----	----	0.1
	Borax	1.3																			

TABLE 3.—*Larvicidal effects of miscellaneous arsenical substances partially adsorbed on charcoal.*

Test No.	Substances used.	Concentration.	Number of larvæ used.	Percentage of dead larvæ in time periods. Cumulative totals.																Weight of larvicide.
				Minutes.								Hours.								
				15	30	45	60	75	90	105	120	2.5	3	4	5	6	7	24	48	72	
		Per cent.																		*g.*
119	Nothing (control)		50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
158	do		50	0	0	0	0	0	0	0	0	0	0	0	0	0	6	6			
4	Arsenic brom phenol	3.0	50	0	0	0	0	0	0	0	0	0	0	0	0	2		4			0.13
106	Atoxyl	0.5	50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	24			0.1
105	Salvarsan	0.2	50	0	0	0	0	0	0	0	0	0	0					0			0.1
104	Salvarsan	0.2	50	0	0	0	0	0	0	0	0	0	0					0			0.1
	Borax	0.4																			
120	Arsenic trioxide	0.5	50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	48			0.1
	Copper carbonate	0.05																			
	Arsenic trioxide	1.0																			
77	Borax	1.0	50	0	2	2	2	2	2	2	2	2	2	2	4			22			0.1
	Lime	1.3																			
88	Arsenic pentoxide	1.0	50	0	0	2	2	2	2	2	2	2	2	2	2	2	2	2			0.1
111	Arsenic pentoxide	0.5	50	0	0	0	0	0	0	0	0	0	0	2	4	6	8	20			0.1
	Aniline	2.0																			
81	Barium arsenite	3.0	50	0	2	2	2	2	2	2	2	2	2	2	2	2		2	2	2	0.13
	Lime	0.5																			
116	Barium arsenite	0.5	50	0	0	0	0	0	0	0	0	0	0	0	0	0		8			0.1
	Ammonium chloride	1.5																			
42	Calcium arsenite	3.0	50	0	0	0	0	0	0	0	0	0	2	2	2	2		2			0.13
	Lime	0.5																			
43	Nickel arsenite	3.0	50	0	0	0	0	0	2	2	2	2	2	2	10	16		48			0.13
	Lime	0.5																			
62	Lead arsenite	7.6	50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4			0.13

58	Arsenic trioxide	2.0	10	0	0	0	0	0	0	0	0	0	0	0	0	10	10	10	(a)	
	Tannic acid	1.0																		
b 87	Arsenic trioxide	1.0	50	0	0	0	0	0	0	0	0	0	0	0	0	0	4	----	0.1	
	Borax-charcoal 1/100																			
121	Potassium arsenite	0.075	50	0	0	0	0	0	0	0	0	0	0	0	0	0	0	----	0.1	
	Copper sulphate	0.125																		
156	Zinc arsenite	2.0	50	0	0	0	0	0	4	4	14	28	70	82	98	98	----	----	0.1	

[a] To cover surface.

[b] No. 87 was a mixture.

NEW OR LITTLE-KNOWN TIPULIDÆ FROM EASTERN ASIA (DIPTERA), X[1]

By Charles P. Alexander

Of Amherst, Massachusetts

THREE PLATES

The Chinese Tipulidæ discussed at this time were chiefly contained in extensive collections made in Szechwan by Mr. Franck and received through the kind interest of Mr. Herbert S. Parish. One additional Chinese species was collected by Prof. Claude R. Kellogg, now at the Massachusetts State College. The more-numerous Japanese crane flies discussed herewith were included in extensive collections made by Messrs. Imanishi, Inomata, Hibi, Kamiya, Machida, Sakaguchi, and Tokunaga. I am very greatly indebted to all of the above-mentioned entomologists for their continued interest in making known the Tipulidæ of China and Japan, and for the privilege of retaining the types in my collection.

TIPULINÆ

DOLICHOPEZA (NESOPEZA) FRANCKI sp. nov. Plate 1, fig. 1; Plate 2, fig. 23.

General coloration of mesonotal præscutum brown, with four brownish black stripes; antennæ relatively short; legs pale brown, the tips of femora, and more narrowly of tibiæ, darkened; tarsi white; wings with a grayish tinge; stigma dark brown, preceded and followed by conspicuous whitish areas; abdominal tergites weakly bicolorous, of the sternites more conspicuously so, the incisures blackened; male hypopygium with the lateral angles of the tergite black, the median ventral point small; inner dististyle heavily blackened.

Male.—Length, about 9 to 9.5 millimeters; wing, 9.5 to 10.

Female.—Length, about 11 to 11.5 millimeters; wing, 10.5 to 11.

Frontal prolongation of head and palpi dark brown. Antennæ with the scape and pedicel light yellow; flagellum dark brown; antennæ relatively short, if bent backward extending to

[1] Contribution from the entomological laboratory, Massachusetts State College.

shortly beyond wing root. Front and anterior vertex light yellow; remainder of head dark brown, in cases with the pale coloration of the anterior vertex continued caudad onto the central portion of the posterior vertex.

Mesonotum with the ground color brown, with four brownish black stripes, the intermediate pair more polished, the lateral pair more obscure; remainder of mesonotum blackish, the postnotal mediotergite dusted with gray. Pleura brownish testaceous, the ventral anepisternum, meron, and ventral sternopleurite blackened, more or less pruinose. Halteres pale, the knobs dark brown. Legs with the coxæ brown; trochanters yellow; femora pale basally, passing into brown, the tips brownish black; tibiæ pale testaceous, the tips narrowly darkened; tarsi snowy white, the proximal ends of basitarsi a trifle more obscure. Wings (Plate 1, fig. 1) with a gray suffusion, the prearcular and costal regions somewhat paler; stigma conspicuous, dark brown, preceded and followed by whitish areas, obliterative area crossing the fork of M more restricted in amount; veins brown. Venation: Rs longer than m-cu; forks of medial field relatively shallow; cell 2d A of moderate width.

Abdominal tergites weakly bicolorous, the bases of the individual segments darkest, the remainder of each paler, with the exception of a narrow caudal darkening; sternites more conspicuously bicolorous, the incisures black, the intermediate portions yellow, the basal darkening more extensive than the narrow apex. Male hypopygium (Plate 2, fig. 23) with the lateral angles of the tergite, 9*t*, heavily blackened, the median region produced caudad into a small ventral point. Outer dististyle, *od*, relatively long. Inner dististyle, *id*, heavily blackened.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 4,500 feet, August 4, 1930 (*Franck*). Allotopotype, female. Paratopotypes, 3 males and females, July 24 to August 20, 1930.

Dolichopeza (*Nesopeza*) *francki* is respectfully dedicated to the collector, to whom I am greatly indebted for many rare Tipulidæ from western China. It is allied to *D.* (*N.*) *albitibia* (Alexander), of Japan, differing in the coloration of the body, legs, and wings, and the details of structure of the male hypopygium, especially of the tergite and inner dististyle.

TIPULA SETICELLULA sp. nov. Plate 1, fig. 2; Plate 2, figs. 24 to 28.

General coloration of thorax black, the abdomen yellow, the outer segments blackened; wings with the outer radial and

medial cells with abundant macrotrichia; r-m connecting with Rs at or shortly before its fork; male hypopygium with the ninth tergite tridentate; eighth sternite with a broad median depressed setiferous lobe.

Male.—Length, about 13 millimeters; wing, 15.

Described from an alcoholic specimen.

Frontal prolongation of head yellowish brown; nasus long and slender; palpi black, the terminal segment paling to yellowish white. Antennæ (male) relatively long, if bent backward extending about to the base of abdomen; scape, pedicel, and first flagellar segment yellow; remaining segments of flagellum weakly bicolorous, the base of each dark brown, the remainder light brown. Head dark, probably with a bloom in dry specimens.

Mesonotum chiefly blackened, the præscutum chiefly covered by the three confluent black stripes; scutal lobes similarly blackened; scutellum obscure yellow, the parascutella dark; postnotum dark. Pleura chiefly dark, the pteropleurite paler; dorsopleural region pale. Halteres pale. Legs with the coxæ yellow, the extreme bases weakly darkened; trochanters yellow; femora yellow, the tips narrowly brownish black, a trifle more extensively so on the forelegs; tibiæ yellowish brown, the tips darkened; tarsi brownish black. Wings (Plate 1, fig. 2) cream-colored, the prearcular and costal regions bright yellow; distal two-thirds of wings gradually darkened, the tip most deeply so; restricted darker areas at origin of Rs and along cord, the latter confluent with the conspicuous stigma; an oblique band of the ground color beyond the stigma, extending from cell R_2 to R_5; less distinct ground areas before stigma and in outer end of cell M; veins brown, flavous in the pale areas. Outer cells of wing with numerous, conspicuous macrotrichia, most extensive in cells R_3 to M_1, with fewer trichia in extreme outer ends of cells 2d M_2 to Cu_1, inclusive. Venation: R_{1+2} entire; Rs long; r-m connecting with Rs at or before the fork of latter; cell 1st M_2 small, pentagonal.

Abdomen yellow, the basal tergites very narrowly trivittate with brown; terminal four segments blackened. Male hypopygium (Plate 2, fig. 24) relatively large, the sutures between tergite, sternite, and basistyle distinct. Ninth tergite (Plate 2, fig. 25) large, not heavily sclerotized, the outer half narrowed, the caudal margin with three lobes, the median lobe smaller and more slender than the laterals, with a sharp median keel on

ventral surface; viewed laterally, the outer lobes are deeper and weakly emarginate. Inner and outer dististyles as shown (Plate 2, fig. 26), the former with a glabrous fingerlike lobe on outer margin at base, the latter cylindrical, with long conspicuous setæ. Gonapophyses (Plate 2, fig. 27) bispinous. Eighth sternite (Plate 2, fig. 28) projecting, the margin broadly notched, bearing a broad, depressed-triangular, median lobe that is densely setiferous; lateral portions of emargination, on either side of median lobe, with a small tuft of five or six stouter setæ.

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Saga, Kyoto Prefecture, altitude 490 feet, August 5, 1928 (*Tokunaga*).

Tipula seticellula is very different from the other Japanese species of *Tipula* having macrotrichia in the outer cells of the wing, differing especially in venation and the structure of the hypopygium. I am uncertain as to the subgeneric position of the present fly. The recent attempt by Edwards to divide the vast genus *Tipula* into subgeneric groups [2] forms a most important contribution to our knowledge of the subject. However, in any extensive treatment of members of this genus, there will long remain species whose strict assignment in subgenera will be uncertain and which must therefore be left unassigned. A similar case exists in *Limnophila* and other large and involved genera. In the past, certain workers have placed such uncertain forms in the typical subgenus, but such a course should be strictly avoided.

TIPULA (TIPULA) OKINAWENSIS sp. nov. Plate 1, fig. 3; Plate 2, fig. 29.

Allied to *yamata;* mesonotum fulvous-orange; wings yellowish brown, the base and costal region more saturated; Rs short, less than m-cu; male hypopygium with the lobes of the tergite obliquely truncated at tips; outer dististyle conspicuously dilated and bearing numerous setæ on lower margin at near midlength; inner dististyle cultriform.

Male.—Length, about 13 millimeters; wing, 16.5.

Frontal prolongation of head light brown; palpi pale brown. Antennæ brown, the basal enlargements of the individual segments darker; flagellar verticils very long. Front light cream-colored; posterior sclerites of head dark brown.

Mesonotal region fulvous-orange. Pleura pale yellow, variegated with light brown on the dorsopleural region; pteropleurite, ventral sternopleurite, and ventral meron slightly darkened.

[2] Ann. & Mag. Nat. Hist. X 8 (1931) 73–82.

Halteres with the stem yellow, the knobs dark brown. Legs with the coxæ and trochanters yellow; femora brownish yellow, the tips narrowly darkened; tibiæ and tarsi brown; hind tibia with two very unequal spurs, the longer being twice the shorter. Wings (Plate 1, fig. 3) with a yellowish brown tinge, the base and costal region more saturated; stigma light brown. A few scattered macrotrichia on vein R_4. Venation: Rs short, about four-fifths m-cu; R_{1+2} unusually short, pale, but entire, gently sinuous; petiole of cell M_1 less than one-half m; cell 2d A very narrow, as in the group.

Abdominal tergites brown, the caudal margins of the individual segments narrowly pale; sternites light yellow. Male hypopygium (Plate 2, fig. 29) with the tergite, 9*t*, bearing two submedian, blackened lobes that are narrowly separated by a linear notch, the tips of the lobes obliquely truncated, provided with small black spines and numerous setæ. Outer dististyle, *od*, with a conspicuous dilation on ventral margin at near midlength, this provided with abundant setæ; beyond the dilation, the style narrows rapidly to a slender point. Inner dististyle, *id*, cultriform, bearing a blackened lobe near base. Lobes of ninth sternite with numerous conspicuous setæ.

Habitat.—Japan (Riukiu Islands).

Holotype, male, Kunjan, Okinawa, altitude 1,000 feet (*S. Sakaguchi*).

"Kunjan-gun, in the northern part of Okinawa; collections made at between 500 and 1,000 feet. The woods are dense and very humid, and along the streams were found very favorable situations for Tipulidae."—S. Sakaguchi.

Tipula okinawensis is allied to *T. yamata* Alexander (Japan) and *T. suensoni* Alexander (eastern China), agreeing in the general coloration and narrow cell 2d A of the wings. In the present species, Rs is unusually short, being less than m-cu in length, while the details of the male hypopygium, especially of the tergite and dististyles, are quite distinct. The members of this restricted group have nearly a score of powerful flattened pale setæ arranged in a comblike series along the dorsal margin of the inner dististyle.

TIPULA (VESTIPLEX) DIVISOTERGATA sp. nov. Plate 2, fig. 30.

Belongs to the *arctica* group; *himalayensis* subgroup; allied to *avicularia;* male hypopygium with the ninth tergite completely divided by pale membrane; a small foot-shaped appendage projecting from beneath each lobe of tergite.

Male.—Length, about 11 to 12 millimeters; wing, 12.5 to 14.

Frontal prolongation of head brownish yellow above, narrowly dark brown on sides; nasus long and conspicuous. Antennæ with the flagellar segments weakly bicolorous, the basal enlargement of each dark brown, the remainder more yellowish brown.

Mesonotal præscutum with a golden-yellow pollen, the three brown stripes narrowly bordered by darker brown, this especially evident on the median vitta. Pleura chiefly yellow or brownish yellow, the ventral sternopleurite a little more darkened. Halteres with the knobs dark brown, their apices conspicuously yellow. Legs with the femora brown, the tips conspicuously blackened, preceded by an obscure yellow ring of approximately equal width. Wings with the pattern about as in *himalayensis,* brown, variegated with creamy areas, especially on the basal half of wing; prearcular and costal regions more yellowish, especially the former.

Abdominal tergites yellow, narrowly trivittate with dark brown, the subterminal segments more uniformly darkened. Male hypopygium (Plate 2, fig. 30) with the tergite, 9*t*, narrowly divided medially by membrane into two halves; caudal margin of tergite with a rounded emargination, the apices of the lobes darker brown, strongly delimited; beneath each lobe on ventral face with a small bilobed structure, more or less foot-shaped, as shown. Outer dististyle, *od*, long and slender, pale throughout. Inner dististyle, *id*, with the blackened margin of the blade smooth. What seems to represent a gonapophysis is a slender, straight rod, weakly expanded at apex into a small head. Ninth sternite, 9*s*, at dorsal outer angle with a small, oval, setiferous lobule. Eighth sternite unarmed.

Habitat.—China (Szechwan).

Holotype, male, Mount Omei, altitude 4,500 feet, July 17, 1929 (*Franck*). Paratopotype, male, July 23, 1929.

Tipula divisotergata seems to find its closest ally among the described species in *T. avicularia* Edwards (Tibet to Sikkim), differing most evidently in the structure of the hypopygium.

CYLINDROTOMINÆ

LIOGMA BREVIPECTEN sp. nov. Plate 1, fig. 4; Plate 2, fig. 31.

Antennal pectinations (male) unusually short and blunt; wings yellowish gray, the wing tip and seams at origin of Rs, along cord and outer end of cell 1st M_2 slightly darker; male hypopygium with the gonapophyses expanded at tips into large, entirely smooth, blades.

Male.—Length, about 13 millimeters; wing, 10.3.

Antennæ (Plate 2, fig. 31) with the scape dark brown, the pedicel and basal two or three flagellar segments passing into obscure yellow; outer flagellar segments brown; flagellar segments with the pectinations unusually short and blunt. Head black, sparsely pruinose.

Mesonotum and pleura black, the dorsopleural region yellow. Halteres pale. Legs with the coxæ brown basally, paler at tips; trochanters yellow; femora yellow, the distal sixth black. Wings (Plate 1, fig. 4) yellowish gray, the prearcular and costal regions more yellowish; stigma small, brown; wing tip and seams at origin of Rs, along cord and outer end of cell 1st M_2 somewhat darker; veins brown, the prearcular and costal veins clearer yellow.

Abdomen black, the outer sternites obscure yellow at base, the margins narrowly blackened. Male hypopygium with the gonapophyses dilated at tips into broadly expanded, entirely smooth blades.

Habitat.—Japan (Honshiu).

Holotype, male, Mount Ohdai, Yamata, altitude 3,250 feet, June 5, 1930 (*S. Sakaguchi*).

The present fly is readily told by the diagnostic features listed above, notably the short antennal pectinations and smooth gonapophyses.

LIOGMA FUSCIPENNIS sp. nov. Plate 1, fig. 5; Plate 2, fig. 32.

Male.—Length, about 13 to 14 millimeters; wing, 10 to 11.5.

Generally similar to *Liogma serraticornis* Alexander (Japan) in most features, differing most evidently in the strongly darkened wings.

Serrations of the flagellum conspicuous (Plate 2, fig. 32). Dorsopleural membrane infuscated; in *serraticornis*, light ocherous. Wings (Plate 1, fig. 5) strongly infuscated, the oval stigma dark brown; cell C still more darkened. Venation: Rs shorter; r-m lost by fusion of R_{4+5} on M_{1+2}, this character constant in all specimens of the type series. In *serraticornis* the wings are conspicuously yellowish, the prearcular and costal regions, together with the stigma, more saturated.

Habitat.—Japan (Honshiu).

Holotype, male, Mount Daisen, Tottori, altitude 3,900 feet, June 7, 1930 (*Hibi*); received through Professor Inomata. Paratopotypes, 2 males, altitude 3,900 to 4,550 feet, June 7, 1930 (*Hibi*).

The relationship of the present fly to *Liogma serraticornis* may perhaps be indicated by a trinomial.

LIMONIINÆ

LIMONIINI

LIMONIA (DICRANOMYIA) TRIFILAMENTOSA sp. nov. Plate 1, fig. 6; Plate 2, fig. 33.

Limonia (*Dicranomyia*) sp. TOKUNAGA, Mem. Coll. Agr. Kyoto Imp. Univ. 10 (1930) 73, 74, 75.

General coloration brownish yellow; antennæ with the basal flagellar segments short-oval; mesonotal præscutum with three darker brown stripes; pleura yellow, longitudinally striped with brown; fore femora with a nearly terminal dark brown ring; wings tinged with brownish yellow; Sc_2 lacking; cell M_2 open by the atrophy of m; male hypopygium with the rostral spines very unequal in length and size, the outer one long and powerful.

Male.—Length, 5.5 to 6 millimeters; wing, 5.5 to 6.

Female.—Length, about 6 to 7 millimeters; wing, 5.5 to 6.

Described from alcoholic specimens.

Rostrum brownish yellow; palpi short, dark brown. Antennæ brown; basal flagellar segments short-oval, the outer segments more elongate-oval; setæ of segments of normal length and size. Head brownish yellow, the center of the vertex darker.

Mesonotal præscutum brownish yellow, with three darker brown stripes, the median stripe narrowly divided by a capillary pale line, most distinct behind; scutal lobes darkened; median region of scutum, and sometimes of scutellum and postnotal mediotergite, restrictedly pale, in other specimens more uniformly darkened. Pleura yellow, striped longitudinally with brown, the dorsal sternopleurite pale. Halteres pale. Legs with the coxæ and trochanters pale; fore femora obscure yellow, with a broad, nearly terminal, dark brown ring; other femora not or only slightly darkened apically; tibiæ and tarsi brownish yellow, the latter only slightly darkened outwardly; claws with five or six teeth, only the outermost of large size. Wings (Plate 1, fig. 6) with a strong brownish yellow tinge, without markings; veins brown. Venation: Sc_1 ending opposite the origin of Rs, Sc_2 lacking; a supernumerary crossvein in cell Sc at near midlength of R; cell M_2 open by atrophy of m, closed only in aberrant individuals; m-cu at or, more usually, a short distance before the fork of M.

Abdomen brown, the sternites somewhat paler. Male hypopygium (Plate 2, fig. 33) with the ninth tergite, 9*t*, transverse, the caudal margin convexly rounded, the median region restrictedly transverse or even feebly concave. Basistyle, *b*, relatively small. Ventral dististyle, *vd*, fleshy, the rostral prolongation weakly chitinized, with two very unequal and widely separated spines. Dorsal dististyle a curved sickle, widest just beyond midlength. Gonapophyses, *g*, with the mesal-apical lobe elongate, a little expanded at tip and with the margin microscopically serrulate.

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Seto, Wakayama-ken, April 15, 1928 (*M. Tokunaga*); marine. Allotopotype, female. Paratopotypes, of both sexes.

In 1928, Dr. Masaaki Tokunaga, while working at the Seto Marine Biological Laboratory, discovered no fewer than three species of marine crane flies, all of which proved to be undescribed. Through the kindness of the collector, two of these species are defined in this report, as *Limonia* (*Dicranomyia*) *trifilamentosa* sp. nov. and *Limonia* (*Idioglochina*) *tokunagai* sp. nov. The third species, *Limonia* (*Dicranomyia*) *monostromia* Tokunaga, has been described in all stages in a beautiful monographic study by the author of the species.[3] The pupa of the present species is most remarkable in the genus *Limonia* in having three-branched breathing horns, whence the specific name, *trifilamentosa*. The discovery of this fly serves to bridge the gap existing between the crane flies of the subtribe Antocharia, having many-branched pronotal breathing horns, and the remaining members of the family, in which this structure is simple. The only other Japanese *Dicranomyia* having cell M_2 open by the atrophy of m is the very different *L.* (*D.*) *immodestoides* (Alexander) with an entirely distinct male hypopygium.

LIMONIA (IDIOGLOCHINA) TOKUNAGAI sp. nov. Plate 1, fig. 7; Plate 2, fig. 34.

? *Gonomyia*, sp. TOKUNAGA, Mem. Coll. Agr. Kyoto Imp. Univ. 10 (1930) 73, 74, 75.

General coloration dark brown to brownish black, probably pruinose in dry specimens; flagellar segments strongly produced; a supernumerary crossvein in cell Sc; male hypopygium

[3] Mem. Coll. Agr. Kyoto Imp. Univ. 10 (1930) 1–127, 17 pls.

with the ventral dististyle a subglobular structure, the rostral prolongation very short and stout, provided with two unequal spines.

Male.—Length, about 5.5 to 7.5 millimeters; wing, 6.2 to 9.

Female.—Length, about 7.5 to 10 millimeters; wing, 6.5 to 10.5.

Described from alcoholic specimens.

Rostrum and palpi brown. Antennæ brownish black throughout; intermediate ten flagellar segments very strongly produced. Head dark brown.

Mesothorax chiefly dark brown, probably pruinose in fresh specimens; scutellum brightened, especially on posterior half. Halteres pale, especially the knobs. Wings (Plate 1, fig. 7) with a strong dusky suffusion, the stigma and vague seams along the cord somewhat darker; veins dark brown. Venation: A supernumerary crossvein in cell Sc at near midlength of vein R; veins of radial field not greatly distorted; cell 1st M_2 closed, as long as or longer than vein M_{1+2} beyond it; m-cu at or before fork of M.

Abdominal tergites black, the sternites somewhat brighter, their caudal margins narrowly darkened; hypopygium black. Male hypopygium (Plate 2, fig. 34) with the tergite, 9*t*, extensive, the caudal margin with a deep and narrow median incision. Basistyle, *b*, elongate, the ventromesal lobe low. Ventral dististyle, *vd*, a subglobular, fleshy structure, the dorsal face deeply incised for the reception of the dorsal dististyle; rostral prolongation very short and obtuse, with two spines, the outer one larger and more powerful than the inner. Gonapophyses narrow, the apical lobe very elongate, obtuse at tip, set with numerous depressed areas.

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Seto, Wakayama-ken, April 15, 1928 (*M. Tokunaga*). Allotopotype, female. Paratopotypes, of both sexes; additional paratopotypes of the smaller summer form, August 15, 1928 (*M. Tokunaga*).

Limonia (*Idioglochina*) *tokunagai* is named after the collector, Dr. Masaaki Tokunaga, student of the marine Diptera of Japan. It is one of three marine crane flies belonging to the genus *Limonia* taken by the collector on the Kii Peninsula in 1928.[4] The present species is very different from the only other described regional *Idioglochina, L.* (*I.*) *kotoshoensis* (Alexander),

[4] Op. cit.

in the nature of serration of the antennal flagellum. It seems highly probable that the larval stage of all of the various species of this subgenus in the Australasian, Oriental, and eastern Palæarctic Regions will be found to occur in a marine habitat. The various members of the group have possibly been derived from some ancestral marine *Dicranomyia* of the general type of *L.* (*D.*) *signipennis* (Coquillett) or *L.* (*D.*) *monostromia* Tokunaga. With the recent discovery [5] by Dr. L. G. Saunders of the marine habitat of *Limonia* (*Geranomyia*) *unicolor* (Haliday), a third subgenus of *Limonia* is thus shown to have marine representatives. The seasonal dimorphism in the present species as briefly noted by Tokunaga [6] is very curious, both sexes of the vernal form being fully one-half larger than the corresponding summer form.

PROANTOCHA QUADRIVITTATA sp. nov. Plate 1, fig. 8.

Male.—Length, about 9 millimeters; wing, 9.

Described from an alcoholic specimen.

Most closely related to *P. spinifer* (Alexander) and *P. serricauda* (Alexander) in the general nature of the vestiture of the legs, differing especially in the thoracic pattern.

Mesonotal præscutum with four distinct brown stripes, the intermediate pair closely approximated for most of their length but well-separated behind; lateral stripes not reaching the suture; scutal lobes each with two dark areas, the more-posterior one larger; postnotal mediotergite with two large, brown, confluent, circular areas on posterior margin. Pleura pale, distinctly marked with brown on the anepisternum and ventral sternopleurite. Fore and middle legs with long abundant delicate setæ; of posterior legs shorter and more reduced, but not spinous; posterior tibiæ with long setæ from enlarged bases, where the setæ are broken, leaving spinous points. Wings as illustrated (Plate 1, fig. 8).

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Saga, Kyoto Prefecture, altitude 490 feet, August 10, 1929 (*Tokunaga*).

Proantocha quadrivittata is intermediate in size between *P. spinifer* and *P. serricauda*, differing from both in the nature of the thoracic pattern. *Proantocha ūyei* Alexander is a very different species, with the vestiture of all legs greatly reduced to spines and short spinous setæ.

[5] Ent. Mo. Mag. 66 (1930) 185–187, figs.
[6] Op. cit. 73.

ANTOCHA SAGANA sp. nov. Plate 1, fig. 9; Plate 2, fig. 35.

General coloration pale, the mesonotal præscutum with a dark brown median stripe; ventral sternopleurite darkened; wings grayish, unmarked; male hypopygium with the ninth tergite evenly covered with microscopic setulæ, its caudal margin gently trilobed; a small setiferous lobule on mesal face of basistyle; both dististyles of nearly equal size and length, weakly expanded into a blade at outer end.

Male.—Length, about 4.2 millimeters; wing, 4.5.

Described from an alcoholic specimen.

Rostrum pale yellow; palpi brown. Antennæ with the space pale, the pedicel and flagellum dark brown; flagellar segments oval, the outer segments becoming more elongate. Head dark brown, the front pale yellow.

Pronotum and mesonotum yellow, with a dark brown median stripe that narrows behind on the præscutum, becoming obsolete some distance before the suture; lateral præscutal stripes lacking; cephalic portions of scutal lobes darkened; posterior half of postnotal mediotergite darkened. Pleura pale, the ventral sternopleurite extensively infuscated. Halteres pale, the outer portion of the stem slightly more infuscated. Legs with the coxæ pale, the fore coxæ more infuscated; trochanters pale; remainder of legs light brown. Wings (Plate 1, fig. 9) with a grayish tinge, the base more whitish; stigma lacking; veins very pale. Macrotrichia on distal two-thirds of last section of R_{4+5} and of M_{1+2} Venation: Sc relatively short, Sc_1 ending about opposite three-fourths the long Rs; m-cu about one-third its length before the fork of M.

Abdomen pale, the tergal incisures restrictedly darkened. Male hypopygium (Plate 2, fig. 35) with the tergite, 9*t*, extensive, its caudal margin gently trilobed, the surface entirely covered by small delicate setulæ. Basistyle, *b*, with a small setiferous lobule on mesal face at base. Both dististyles relatively elongate, of generally equal size and shape, being slightly arcuate, the distal ends weakly expanded into a blade; outer style, *od*, glabrous, the inner style, *id*, with long, coarse, chiefly marginal setæ.

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Saga, Kyoto Prefecture, altitude 490 feet, April 15, 1928 (*Tokunaga*).

Antocha sagana is readily told from all other regional species of the genus by the structure of the male hypopygium, especially of the dististyles.

DICRANOPTYCHA MACHIDANA sp. nov. Plate 1, fig. 10; Plate 3, fig. 36.

General coloration dark brown, including the abdomen; halteres yellow; legs yellow, the tips of femora narrowly but abruptly blackened; extreme base and tip of tibia more narrowly blackened; wings brownish yellow, the veins dark brown, those in the costal and prearcular areas more yellowish; male hypopygium with the lateral lobes of the tergite appearing as pale spatulate blades; inner dististyle terminating in a yellow apical point.

Male.—Length, about 8.5 millimeters; wing, 10.5.

Rostrum and palpi dark brown. Antennæ with the scape dark brown above, paler beneath; pedicel blackened, paler apically; flagellum broken. Head dark brown, crushed in the type.

Thorax chiefly dark brown, badly crushed in the type, the pleural sclerites surrounding the wing root paler. Halteres yellow. Legs with the coxæ and trochanters yellow, the fore coxæ darker; femora yellow, the tips narrowly but conspicuously blackened; tibiæ yellow, the extreme base very narrowly, the tip a trifle more broadly blackened; basitarsi yellow, the tips and remainder of tarsi brownish black. Wings (Plate 1, fig. 10) brownish yellow, the costal and prearcular regions clearer yellow; veins dark brown, C, Sc, R, and the prearcular veins more yellowish. Venation: Rs long, fully three times the basal section of R_{4+5}.

Abdomen brownish black, the hypopygium a little paler. Male hypopygium (Plate 3, fig. 36) with the lateral lobes of tergite, 9*t*, appearing as pale, spatulate blades, their apices evenly rounded. Outer dististyle, *od*, slender, the distal third blackened and set with appressed to suberect spines. Inner dististyle, *id*, elongate, narrowed outwardly and terminating in a chitinized yellow apical point.

Habitat.—Japan (Honshiu).

Holotype, male, Mount Hakutai, Chichibu, August 25, 1930 (*J. Machida*).

I take great pleasure in naming this distinct fly after the collector, my friend Dr. Jiro Machida, to whom I am very greatly indebted for many interesting Japanese Tipulidæ. The species is most similar in its general features to *Dicranoptycha geniculata* Alexander (Formosa), the pattern of the legs being very similar in the two flies. It differs very conspicuously in the wing markings, venation, and structure of the male hypopygium. The present species is very different from the other members of the genus known from the major islands of Japan.

HELIUS (HELIUS) PLUTO sp. nov. Plate 1, fig. 11; Plate 3, fig. 37.

Allied to *H.* (*H.*) *tenuirostris;* general coloration of thorax brownish black; legs brownish black; wings with a faint brown tinge, the base and costal region light yellow; male hypopygium with the lateral lobe of tergite terminating in a slender acute spine; apex of inner dististyle long and slender.

Male.—Length, excluding rostrum, about 8 millimeters; wing, 8; rostrum, 2.7.

Female.—Length, excluding rostrum, about 9 millimeters; wing, 8; rostrum, 2.8.

Rostrum black, elongate, as shown by the measurements, being approximately as long as the combined head and thorax; palpi black. Antennæ black throughout. Head dark gray; anterior vertex relatively narrow, approximately as wide as the diameter of the base of rostrum, provided with numerous erect black setæ; ommatidia fine.

Pronotum and cervical sclerites dark brown, the former paling to obscure yellow on sides. Mesonotum chiefly brownish black, the humeral region of præscutum vaguely brightened. Pleura chiefly brownish black, the dorsal pteropleurite paler. Halteres light brown, the base of stem paler. Legs with the fore coxæ dark brown, the remaining coxæ brownish yellow; trochanters light yellow; remainder of legs brownish black, the femoral bases obscure yellow, most extensive on posterior legs. Wings (Plate 1, fig. 11) with a faint brownish tinge, the base and costal region light yellow; stigma oval, darker brown; a dusky seam adjoining vein Cu_1; veins dark brown. Venation: Sc_1 ending shortly before the fork of Rs, Sc_2 at the extreme tip and more heavily sclerotized; branches of Rs gently diverging; m-cu close to fork of M.

Abdominal tergites feebly dimidiate, light brown basally, the caudal half black, the amount of the latter increasing on the outer segments. In female the tergites are more uniformly darkened. Male hypopygium (Plate 3, fig. 37) with the lateral lobes of the tergite, 9*t*, terminating in a slender acute spine. Basistyle, *b*, with the mesal face densely set with setæ. Outer dististyle, *od*, a straight blackened rod, the apex very weakly bispinous. Apex of inner dististyle, *id*, very long and narrow.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 3,000 feet, July 25, 1930 (*Franck*). Allotopotype, female, July 23, 1930.

Helius (*Helius*) *pluto* is most nearly allied to *H.* (*H.*) *rufithorax* Alexander (Formosa) and *H.* (*H.*) *tenuirostris* Alexander

(Japan), differing from the former in the coloration of the thorax and from the latter in the larger size and general coloration. The distinctive features of the male hypopygium of the present fly lie in the shape of the lateral lobes of the tergite and the long, slender tip of the inner dististyle. It should be noted that the abdomen of the male of *tenuirostris* has the individual segments dimidiate, yellow on the basal half, the apices abruptly blackened. This character was wrongly described by the writer in the original definition of this species.

HEXATOMINI

POLYMERA PARVICORNIS sp. nov. Plate 1, fig. 12; Plate 3, fig. 38.

General coloration pale brown; antennæ short; wings tinged with brown; no stigmal area; cell M_1 present; male hypopygium with the outer dististyle a simple rod, the tip obtuse, with an acute subapical spine.

Male.—Length, about 4.5 millimeters; wing, 4.6.

Described from an alcoholic specimen.

Rostrum short, pale yellow; palpi relatively long, especially the two, slender, terminal segments, the whole about one-third longer than the head and some two-thirds as long as the antenna. Antennæ with the scape and pedicel yellow, the flagellum dark brown; antennæ (male) short and unmodified, about as long as the thorax alone; antennæ apparently only 15-segmented, the segments subcylindrical; terminal segment elongate, nearly as long as the preceding two segments combined and apparently formed by the fusion of segments. Head brown, the front and broad orbits yellowish.

Pronotum brown. Mesonotum brownish yellow, the præscutum with a double yellow area on disk, narrowly margined with delicate brown lines, the dividing mark on the median area of the sclerite; extreme cephalic-median portion of præscutum darkened; mesal edges of scutal lobes similarly dark-margined; a narrow median yellow vitta extending from the suture to mid-length of the postnotal mediotergite, more interrupted on the scutellum; pseudosutural foveæ very small, pale. Pleura yellow, the dorsal sclerites more darkened. Halteres pale, the bases of the knobs a little darker. Legs with the coxæ and trochanters yellow; remainder of legs pale brown; segments with relatively long, conspicuous setæ; claws small. Wings (Plate 1, fig. 12) tinged with brown; no stigmal area or other markings; veins pale brown, with long conspicuous black verticils on almost all longitudinal veins, lacking on basal half of first section of Cu_1

and 2d A. Venation: Sc_1 ending shortly before fork of Rs, Sc_2 a short distance from its tip; R_{2+3+4} longer than R_{2+3}; R_2 faint; R_{1+2} a little longer than R_{2+3+4}; cell M_1 present; m-cu close to fork of M; anterior arculus present.

Abdomen brown, the sternites paler. Male hypopygium (Plate 3, fig. 38) with the outer dististyle, *od*, a simple, nearly straight rod, the apex bluntly obtuse, with a small subapical appressed spine.

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Kibune, Kyoto Prefecture, altitude 750 feet, at light, June 1, 1930 (*Tokunaga*).

The generic reference of this curious fly must be considered as being somewhat provisional, since the species differs so conspicuously from all hitherto-described members of the genus in the short male antennæ. I at first considered the fly to belong to *Troglophila* Brunetti, where it would be equally isolated by the brevity and structure of the antennæ. The venation and trichiation, together with the basic plan of structure of the male hypopygium, are so much as in *Polymera* that I am venturing to refer the species to this essentially Neotropical genus of Tipulidæ. It would appear that *Troglophila* Brunetti and *Polymera* Wiedemann are more closely allied than was hitherto considered. The ecological conditions at Kibune have been described and figured by Iwata, in an entomological survey of the upper Kamogawa River, with special reference to trichopterous larvæ and pupæ.[7]

LIMNOPHILA (ELÆOPHILA) SERRULATA sp. nov. Plate 1, fig. 13; Plate 3, fig. 39.

Size small (wing, male, 5 millimeters); legs yellow, the femora with a very slightly darker brown ring immediately before tips; wings (male) suddenly widest opposite end of vein 2d A; whitish, with an abundant dotted and spotted brown pattern; male hypopygium with the apex of the outer dististyle microscopically serrulate.

Male.—Length, about 4.2 millimeters; wing, 5.

Rostrum and palpi black. Antennæ with the scape and pedicel black; basal segments of flagellum pale brown, the outer segments somewhat darker; basal flagellar segments somewhat swollen on ventral face. Head blackish, the anterior vertex and narrow orbits more grayish.

[7] Bull. Biogeograph. Soc. Japan No. 1, 2 (1930) 1–20, 5 pls.

Mesonotum yellowish gray, conspicuously variegated with dark brown stripes and dashes, including two elongate intermediate stripes that are obsolete in front and interrupted just before suture; lateral stripes bending mesad and becoming confluent at anterior ends with the intermediate stripes; extreme lateral margins of præscutum, behind the pseudosutural foveæ, dark brown; a series of three or four brown punctigerous spots on posterior interspaces; pseudosutural foveæ black; posterior sclerites of mesonotum chiefly blackened. Pleura black, sparsely interspersed with more-grayish areas. Halteres yellow, the knobs brownish black. Legs with the coxæ and trochanters blackish, the latter slightly brightened at bases; remainder of legs yellow, the femora with a slightly darker brown subterminal ring. Wings (Plate 1, fig. 13) with the ground color whitish, the prearcular region light yellow; an abundant dotted and spotted brown pattern, including about six major costal areas, the second at level of origin of Rs entirely crossing the wing as a narrow fascia; third area at end of Sc, fourth at tip of R_{1+2}, the two latter converging behind to form the largest area on disk, lying over the anterior cord; fifth and sixth costal areas at ends of veins R_3 and R_4, respectively; wing apex as far caudad as cubital field, narrowly bordered by brown; interspaces with numerous small brown dots, mostly contiguous to the veins but with some in centers of cells; veins pale, darker in the infuscated areas. Wing (male) abruptly widest opposite end of vein 2d A. Costal fringe of moderate length only. Venation: Supernumerary crossvein in cell M lying immediately distad of level of origin of Rs.

Abdominal tergites black, the sternites paler medially. Male hypopygium (Plate 3, fig. 39) with the basistyle, *b*, produced mesally into an obtuse lobe. Outer dististyle, *od*, with the outer spine relatively blunt, placed at beyond midlength of style; apex of style microscopically serrulate. Inner dististyle relatively narrow.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 4,000 feet, July 28, 1930 (*Franck*).

Of the regional species in eastern Asia, *Limnophila* (*Elæophila*) *serrulata* most resembles *L.* (*E.*) *dietziana* Alexander (Japan) in its small size and abundantly spotted wings, differing conspicuously in the lack of black femoral tips and the

serrulate outer dististyle of the male hypopygium. In the latter character, the present fly likewise differs from the larger *L.* (*E.*) *suensoni* Alexander (eastern China). There seems to be no possibility of our further refusing to use the subgeneric term *Elæophila* Rondani, in place of the more familiar and generally used *Ephelia* Schiner.

LIMNOPHILA IMANISHII sp. nov. Plate 3, fig. 40.

Subapterous, at least in the female sex; antennæ 13-segmented the flagellar segments short-oval; terminal segment elongate, evidently resulting from the fusion of two segments.

Female.—Length, about 8 millimeters; wing, 1.3.

Rostrum brown; palpi with the terminal segment considerably longer than the third. Antennæ (Plate 3, fig. 40) 13-segmented, brown throughout; pedicel not conspicuously elongate, about one-half the length of the scape; flagellar segments short-oval to subglobular; outer segments becoming more elongate, the penultimate with an apical ring of verticils in addition to the basal ring; terminal segment elongate, equal to the preceding two taken together, with an incomplete suture, indicating a fusion of segments. Head brown.

Mesonotum relatively small, brown. Halteres pale, small, about one-half the length of the wings, the club linear. Legs with the middle and hind coxæ elongate, dark brown; trochanters brown; legs relatively short and stout, brownish yellow; tibial spurs elongate, setiferous; tarsal claws smooth. Wings greatly reduced, stenopterous, uniformly yellowish brown; no clearly defined venation except beyond the cord where indicated by macrotrichia; cell M_1 present. Macrotrichia on C and R for almost their entire length, on other veins only beyond the level of the cord, on R_4, M_1, M_2, M_3, and M_4.

Abdomen dark brown, the lateral line somewhat darker. Ovipositor with the tergal valves elongate, slender, gently upcurved; sternal valves compressed.

Habitat.—Japan (Honshiu).

Holotype, alcoholic female, Bogaya, Toyama, Japanese Alps, altitude 3,900 feet, on snow (*Imanishi*).

Limnophila imanishii is named in honor of the collector, Mr. K. Imanishi, who has taken many rare Tipulidæ in the Japanese mountains. The species cannot be associated with any of those previously described. I place the fly in *Limnophila* in the broad definition of the genus only. Nearly apterous females of hexatomine and pediciine Tipulidæ are very difficult to place ac-

curately as to genus, but the reference of the present fly to the Hexatomini seems justified by the glabrous eyes.

ERIOCERA NIGROTROCHANTERATA sp. nov. Plate 1, fig. 14.

Male.—Length, about 13 millimeters; wing, 16 by 5.

Belongs to the *spinosa* group; closely related to *E. issikii* Alexander (Honshiu) in the short antennæ of the male sex.

Rostrum and palpi black. Antennæ black throughout, 7-segmented; terminal segment small; first flagellar segment longer and slenderer than in *issikii,* being approximately one-third longer than the second palpal segment; in *issikii* the second palpal segment is subequal to or even a little longer than the first flagellar segment. Mesonotal præscutum with four dark stripes, the intermediate pair narrow; in *issikii,* with three such stripes, the broad median stripe being entire; scutellum black. Halteres black, only the base of stem pale. Hind legs with the black femoral tips narrow, including about the distal fourth; in *issikii* broad, including the distal half or slightly more. Wings (Plate 1, fig. 14) somewhat shorter and broader; veins of radial and medial fields with broad, conspicuous, brown seams.

Habitat.—Japan (Honshiu).

Holotype, male, Mount Ohdai, Yamato, altitude 2,600 feet, June 5, 1930 (*Sakaguchi*).

ERIOCERA KELLOGGI sp. nov. Plate 1, fig. 15.

General coloration black, the thoracic stripes and bases of abdominal tergites more plumbeous, glabrous; femora and halteres black; wings brown, the base paler than the apical third, with a very vague, more yellowish crossband before the cord; macrotrichia of costa much reduced in size and number.

Male.—Length, about 16 millimeters; wing, 15.5.

Rostrum and palpi black. Antennæ 7-segmented; scape and pedicel dark brown; flagellum yellowish brown, the outer segments again darkened. Head black, the vertical tubercle relatively low.

Mesonotal præscutum grayish brown, with four subnitidous, plumbeous black stripes that are narrowly separated by lines of opaque black; posterior sclerites of mesonotum dull black. Pleura black. Halteres black. Legs with the coxæ and trochanters black; femora brownish black; tibiæ brown, the tips narrowly darkened; tarsi dark brown. Wings (Plate 1, fig. 15) very vaguely cross-banded, the basal two-thirds obscure brownish yellow, the apical third infuscated, preceded by a scarcely defined clearer yellow crossband before the cord, most extensive

in the radial and medial fields, more or less narrowed to a point posteriorly at m-cu; costal region more intense orange-yellow; veins yellow, somewhat darker in the infumed apical portion. Macrotrichia of veins very small and reduced, very evidently so on costa between h and the level of R_2, the trichia becoming more abundant on costa beyond the latter point to the wing tip. Venation: Tip of Sc_1 atrophied about opposite R_2; cell M_1 present; m-cu at near midlength of cell 1st M_2.

Abdominal tergites polished leaden blue-black, the caudal margins opaque black, the latter including about the apical third of the intermediate segments; sternites and hypopygium black.

Habitat.—China (Fukien).

Holotype, male, Foochow (*Kellogg*).

I take great pleasure in naming this species in honor of the collector, Prof. Claude R. Kellogg, of the Massachusetts State College. By Edwards's key to the Old World species of *Eriocera*[8] the present fly runs to couplet 71, disagreeing in the combination of leaden basal crossbands on the abdominal tergites, in conjunction with the presence of cell M_1 of the wings. It is closely allied to *E. muiri* Alexander, of Macao,[9] which differs in the clearly defined yellow crossband of the wing and the more-abundant macrotrichia of the veins of the radial and medial fields. The great reduction in number and size of the macrotrichia of the costa in *E. mesopyrrha* (Wiedemann), *E. prælata* Alexander, *E. muiri* Alexander, the present species, and probably still others in the Oriental and Eastern Palæarctic faunal regions is noteworthy.

ERIOCERA ARROGANS Alexander.

Eriocera arrogans ALEXANDER, Proc. U. S. Nat. Mus. 72, art. 2 (1927) 5–6.

Described from a unique female specimen taken at Mount Omei, Szechwan, China, by Rev. David C. Graham. Three additional specimens were taken at Kwanhsien, Szechwan, altitude 3,000 feet, July 18 to 24, 1930; altitude 4,000 feet, August 12, 1930 (*Franck*).

The males are much smaller than the females (length, 8 to 8.5 millimeters; wing, 9.2 to 9.8) and have the wings almost uniformly blackened, without the conspicuous pale centers to the cells found in the female sex. By Edwards's key to the

[8] Ann. & Mag. Nat. Hist. IX 8 (1921) 70–78.

[9] Proc. Hawaiian Ent. Soc. 5 (1923) 255.

Old World species of *Eriocera,*[10] the fly runs to *E. unicolor* de Meijere,[11] differing in the small size and details of coloration. The specimens from Formosa referred to *unicolor* by Edwards [12] were later described as a new species, *E. lygropis* Alexander.[13]

ERIOCERA CÆSAREA Alexander.

Eriocera cæsarea ALEXANDER, Philip. Journ. Sci. 44 (1931) 358–359.

The fly was described from a unique male specimen, taken at Kwanhsien, Szechwan, China, by Rev. David C. Graham. An additional male from the type locality (altitude 2,800 feet, July 28, 1930, *Franck*) agrees closely with the type, differing in a few details only. Wings with indications of a very broad, scarcely evident, yellowish brightening before the cord. Venation: Cell 1st M_2 slightly more elongate, with m-cu at midlength of its lower face.

ERIOCERA KAMIYAI sp. nov. Plate 1, fig. 16.

General coloration velvety black, including the head, thorax, and abdomen; mesonotal præscutum with four polished black stripes; halteres entirely black; legs black, the femora chiefly yellow, the tips blackened; wings with the ground color cream-yellow, very heavily patterned with brown, including cells C and Sc and broad seams at origin of Rs, along cord and outer end of cell 1st M_2; vein R_{2+3+4} short.

Male.—Length, about 12 millimeters; wing, 11.3.

Rostrum and palpi black. Antennæ black throughout, the terminal segments broken. Head velvety black; vertical tubercle conspicuous.

Mesonotal præscutum velvety black, with four polished black stripes that are little evident against the ground color; remainder of mesonotum and pleura velvety black. Halteres short, black throughout. Legs with the coxæ and trochanters black; femora obscure yellow, the tips blackened, narrowest on the posterior legs; tibiæ and tarsi black. Wings (Plate 1, fig. 16) with the ground color cream-yellow but almost concealed by an extensive dark brown pattern, chiefly evident in the cells before and beyond the cord; cells C and Sc uniformly darkened; very extensive dark clouds at origin of Rs, along cord, at stigma, and outer end of cell 1st M_2; wing apex margined with dusky;

[10] Loc. cit.
[11] Fauna Simalurensis—Diptera, Tijd. voor Ent. 58 (1915) 12–13.
[12] Ann. & Mag. Nat. Hist. VIII 18 (1916) 253.
[13] Ann. Ent. Soc. Am. 13 (1920) 259–260.

anal cells more uniformly grayish brown; veins black. Venation: R_{2+3+4} short, a little more than one-half the basal section of R_5; R_2 less than R_{1+2}, placed shortly before midlength of the anterior branch of Rs; cell M_1 lacking; m-cu just beyond one-third the length of the long cell 1st M_2; distal section of Cu_1 short.

Abdomen velvety black, including the hypopygium.

Habitat.—Japan (Honshiu).

Holotype, male, Kamikochi, Shinano, Japanese Alps, July 29, 1930 (*Kamiya*); through Dr. Jiro Machida.

Eriocera kamiyai is named in honor of the collector, Mr. K. Kamiya. The species is most nearly allied to *E. fulvibasis* Alexander, *E. longifurca* Alexander, and *E. subrectangularis* Alexander, all of Japan, differing in the velvety black coloration of the body and halteres, and the unusually heavy pattern of the wings, cells C and Sc being uniformly darkened.

ATARBA (ATARBODES) BIPUNCTULATA sp. nov. Plate 1, fig. 17; Plate 3, fig. 41.

General coloration pale yellow, each side of pleura with two dark brown spots, placed on the pteropleurite and the pleurotergite; halteres and legs yellow; wings with a strong yellow tinge; Sc relatively long, Sc_1 ending opposite or beyond midlength of Rs; male hypopygium with the outer dististyle bearing spines on the distal fifth or less; gonapophyses appearing as short, macelike structures.

Male.—Length, about 4.5 millimeters; wing, 5.

Rostrum whitish yellow; palpi with the basal segment yellow, the remainder black. Antennæ with the scape and pedicel brown, the flagellum brownish black; antennæ (male) short, the segments with long conspicuous verticils that much exceed the segments. Head light yellow.

Mesonotum pale yellow, the præscutum a trifle more brownish yellow in front but otherwise immaculate; scutellum testaceous. Pleura pale yellow, with a conspicuous brownish black spot on pteropleurite, and another, slightly more elongate, paler brown area on the pleurotergite. Halteres pale yellow. Legs with the coxæ and trochanters yellow; remainder of legs yellow, without infuscation on femora or tibiæ; outer tarsal segments a little darker. Wings (Plate 1, fig. 17) with a strong yellow tinge, the veins deeper yellow, some of them very pale and indistinct; veins beyond cord with conspicuous macrotrichia. Venation: Sc relatively long, Sc_1 ending about opposite or just beyond midlength of Rs.

Abdomen yellow, the subterminal segments brown. Male hypopygium (Plate 3, fig. 41) with the basistyle, *b*, unarmed with a lobe on mesal face at apex. Outer dististyle, *od*, with the apex blackened and here provided with many subappressed spines, the latter not including more than the distal fifth of style; inner dististyle longer than the outer, more expanded at near midlength. Ædeagus long. Gonapophyses, *g*, appearing as short, powerful, macelike structures, the apex of each set with many short spines.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 3,000 feet, July 19, 1930 (*Franck*).

By my key to the species of *Atarba* of the eastern Palæarctic Region [14] the present species runs to *A. leptoxantha* Alexander (Formosa) and *A. issikiana* Alexander (Formosa). It differs very conspicuously in the structure of the male hypopygium, notably the toothing of the outer dististyle and the peculiar macelike structure of the gonapophyses.

ERIOPTERINI

CHIONEA NIPPONICA sp. nov. Plate 3, fig. 42.

Allied to *C. araneoides;* male hypopygium with the dististyle terminating in spinous setæ; gonapophyses appearing as pale flattened plates.

Male.—Length, about 5.5 millimeters.

Female.—Length, about 6 millimeters.

Closely allied to *C. araneoides* Dalman (Plate 3, fig. 43), differing especially in the structure of the male hypopygium.

Antennæ, male, 9- to 10-segmented; female, 10-segmented. Male hypopygium (Plate 3, fig. 42) with the basistyle, *b*, stout, the mesal face at proximal end with an oval area set with long erect setæ. Outer lobe of dististyle a small stout blackened structure, weakly bidentate; main arm of style long and slender, at base on inner margin with a blunt tooth; apex of style, *d*, with several spinous setæ, outer face of style with the setæ greatly reduced in number. Gonapophyses, *g*, appearing as pale flattened plates that converge toward the ædeagus. The paratype has the group of setæ on mesal face of basistyle less restricted in area.

Chionea araneoides (Plate 3, fig. 43) lacks a delimited setiferous area on mesal face of basistyle, *b*. Outer lobe of disti-

[14] Philip. Journ. Sci. 42 (1930) 526.

style more acutely produced into a blackened tooth; main arm with only a slight convexity at base, this with conspicuous setæ; surface of style, *d*, with long conspicuous setæ, those at apex not stout and spinous. Gonapophyses, *g*, appearing as blackened toothlike structures.

Habitat.—Japan (Honshiu).

Holotype, male, Mount Tsurugi, Toyama, Japanese Alps, altitude 8,775 feet, on snow, October 20, 1928 (*Imanishi*). Allotopotype, female, with the type; Tokunaga No. 10. Paratopotype, female, October 16, 1928; Tokunaga No. 14. Paratype, male, Seki, Niigata, altitude 2,925 feet, January 11, 1923 (*ex Tokunaga*).

Chionea nipponica is readily distinguished from the allied *C. araneoides* Dalman (Europe) by the structure of the male hypopygium. This is very probably the species recorded by Matsumura [15] as *Chionea araneoides*, taken indoors during November at Sapporo, Hokkaido. The present records are the most southerly for any member of the genus *Chionea* in eastern Asia.

GONOMYIA (GONOMYIA) SUBCOGNATELLA sp. nov. Plate 1, fig. 18; Plate 3, fig. 44.

Belongs to the *cognatella* group; rostrum and palpi black; antennæ with the two basal segments yellow, the remainder black; thoracic pleura dark brown, with a broad, yellowish white, longitudinal stripe; wings with a strong brownish yellow tinge, the stigma slightly darker; R_{2+3+4} arcuated; abdominal segments dimidiate, dark brown, ringed caudally with yellow; male hypopygium with the outer dististyle a slender, setiferous rod; inner dististyle with the inner arm terminating in a very long, black spine.

Male.—Length, about 3.5 millimeters; wing, 4.2.

Female.—Length, about 4.5 millimeters; wing, 4.6.

Rostrum and palpi brownish black. Antennæ with the scape and pedicel light yellow; flagellum black; flagellar segments long-oval, clothed with a dense white pubescence. Head light yellow, the center of vertex infuscated.

Pronotum and anterior lateral pretergites light yellow. Mesonotal præscutum brownish gray; pseudosutural foveæ dark reddish; scutellum yellow, more infuscated in female. Pleura dark brown, with a broad yellowish white longitudinal stripe extending from and including the fore coxæ, occupying the dorsal sternopleurite, ventral pteropleurite, and most of the meral

[15] Konchu Bunruigaku 2 (1915) 63, pl. 2, fig. 15.

region. Halteres pale, the knobs weakly darkened. Legs with the fore coxæ very pale yellow, the remaining coxæ and all trochanters brownish yellow; remainder of legs pale brown, the femoral bases paler. Wings (Plate 1, fig. 18) with a strong brownish yellow tinge, the prearcular and costal regions clear light yellow; stigma a little darker than the ground color; a scarcely indicated darkened seam along cord; veins light brown, brighter in the yellow areas. Venation: Sc_1 ending opposite or just beyond origin of Rs, Sc_2 at its extreme tip, not apparent in the type female; R_{2+3+4} rather strongly arcuated, less so in female; cell 1st M_2 open by atrophy of basal section of M_3; m-cu close to fork of M.

Abdominal tergites dark brown, conspicuously ringed caudally with yellow; sternites paler brown, the caudal margins yellow. Male hypopygium (Plate 3, fig. 44) with the outer dististyle, *od*, slender, simple, with numerous setæ, especially along outer face. Inner dististyle, *id*, with the inner arm, *ia*, slender, terminating in a very long, slender, black spine.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 3,000 feet, July 18, 1930 (*Franck*). Allotype, female, Chengtu, altitude 1,700 feet, May 21, 1930 (*Franck*).

The possibility exists that the female allotype may not be conspecific with the type male. *Gonomyia* (*Gonomyia*) *subcognatella* differs from *G.* (*G.*) *aperta* Brunetti (British India) in the coloration of the pleura and abdomen and in the details of venation. These are the only two members of this characteristic group of the genus known to me in Asia. Of the numerous Nearctic species of the group, the present fly has an outer dististyle that is most like that of *G.* (*G.*) *kansensis* Alexander, while the simple inner arm of the inner dististyle is somewhat like that found in *G.* (*G.*) *florens* Alexander. The unusually long spine of this arm of the dististyle is distinctive of the present species.

ORMOSIA TOKUNAGAI sp. nov. Plate 1, fig. 19; Plate 3, fig. 45.

General coloration brown; antennæ short; cell M_2 open by the atrophy of the outer section of M_3; male hypopygium with both dististyles small; gonapophyses appearing as slender curved blades, each terminating in a blackened spine, the apophyses subtended by a long, slender, pale, lateral spine.

Male.—Length, about 4.5 millimeters; wing, 4.4.

Described from an alcoholic specimen.

Rostrum brownish yellow; palpi light brown. Antennæ (male) short, pale throughout; flagellar segments oval. Head brown, darker medially above.

Mesonotal præscutum chiefly light brown, the posterior sclerites of the mesonotum, together with the pleura, darker brown. Halteres yellow. Legs with the coxæ brown; trochanters yellowish brown; legs chiefly yellow or brownish yellow. Wings (Plate 1, fig. 19) yellowish gray, the stigmal region somewhat darker; veins slightly darker than the ground color, rather stout. Venation: Cell M_2 open by the atrophy of basal section of M_3; m-cu shortly before fork of M; vein 2d A gently sinuous on outer half.

Abdomen light brown, the caudal margins of the segments narrowly darker. Male hypopygium (Plate 3, fig. 45) with the dististyle, *d*, almost as in *O. takahashii* and *O. takeuchii*. Gonapophyses, *g*, very different, appearing as slender, curved blades, each terminating in a blackened spine; a long, slender, pale, lateral spine arising near the base of the apophysis.

Habitat.—Japan (Honshiu).

Holotype, alcoholic male, Kibune, Kyoto Prefecture, altitude 750 feet, October 2, 1929 (*Tokunaga*).

This interesting *Ormosia* is named in honor of the collector, Dr. Masaaki Tokunaga. Allied to *Ormosia takahashii* Alexander and *O. takeuchii* Alexander, both of Japan, differing conspicuously in the structure of the male hypopygium.

ERIOPTERA (ILISIA) SUBAREOLATA sp. nov. Plate 1, fig. 20; Plate 3, fig. 46.

Belongs to the *areolata* group; allied to *sachalina;* legs yellow, only the terminal tarsal segments darkened; wings brownish yellow, the outer costal region darker; veins along cord infuscated; male hypopygium with the lateral arms of gonapophyses relatively stout.

Male.—Length, about 3.5 millimeters; wing, 4.

Rostrum brown; palpi black. Antennæ broken. Head pale brown, the anterior vertex light gray.

Mesonotum light brown, sparsely dusted with gray, the præscutum not variegated; pseudosutural foveæ and tuberculate pits chestnut-brown. Pleura light grayish brown. Halteres broken. Legs with the coxæ and trochanters testaceous yellow; remainder of legs yellow, the terminal tarsal segments darker. Wings (Plate 1, fig. 20) brownish yellow, the outer costal region more saturated; veins pale brown, the cord and outer end of

cell 1st M_2 darker brown. Venation: Sc_1 ending opposite R_2; cell 1st M_2 small; m-cu about one-third its length before the fork of M; veins M_3 and M_4 deflected strongly cephalad at margin, vein Cu_1 less strongly so.

Abdominal tergites brown, the caudal margins of the segments pale yellow; sternites more uniformly yellow; hypopygium yellow. Male hypopygium (Plate 3, fig. 46) with the outer arm of the outer dististyle, *d*, only feebly blackened; intermediate arm simple. Lateral arms of gonapophyses, *g*, stouter than in *sachalina*.

Habitat.—China (Szechwan).

Holotype, male, Chengtu, altitude 1,700 feet, April 17, 1930 (*Franck*).

Erioptera (*Ilisia*) *subareolata* is most closely allied to *E.* (*I.*) *sachalina* Alexander (Saghalien), differing especially in the pattern of the legs and the slightly different male hypopygium, notably the feebly blackened outer arm of the outer dististyle and the stout lateral arms of the gonapophyses.

MOLOPHILUS ALBIREO sp. nov. Plate 1, fig. 21; Plate 3, fig. 47.

Belongs to the *gracilis* group and subgroup; general coloration dull black; antennæ (male) short; knobs of halteres dark brown; legs brownish black; wings with a strong blackish suffusion; costal fringe (male) conspicuous; male hypopygium with the ventral lobe of basistyle well developed, the dorsal lobe small, unblackened; both dististyles small and compressed.

Male.—Length, about 3.5 millimeters; wing, 4.

Rostrum and palpi black. Antennæ (male) short, black throughout; flagellar segments subcylindrical. Head dull black.

Anterior lateral pretergites light yellow. Mesonotum dull black, very sparsely pruinose; humeral region restrictedly brightened; pseudosutural foveæ and tuberculate pits black. Pleura dull black, sparsely pruinose, the dorsopleural membrane dark. Halteres brown, the knobs dark brown. Legs with the coxæ brownish black; trochanters brown; remainder of legs brownish black. Wings (Plate 1, fig. 21) with a strong blackish suffusion; veins and macrotrichia black. Costal fringe (male) relatively long and conspicuous, especially on distal half of wing. Venation: R_2 and r-m nearly in transverse alignment; vein 2d A ending about opposite the caudal end of m-cu.

Abdomen black. Male hypopygium (Plate 3, fig. 47) with the ventral lobe of basistyle, *b*, elongate, extending caudad be-

yond level of dististyle, armed with retrorse setæ; dorsal lobe small, unblackened. Dorsal dististyle, *dd*, a flattened blade; ventral dististyle, *vd*, with the inner angle prolonged.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 4,000 feet, August 16, 1930 (*Franck*).

Molophilus albireo belongs to a small aggregation of species with the lobes of the basistyle of the male hypopygium feebly developed and having no spinous points. The structure of the dististyles, together with the coloration, especially the dark halteres, readily suffice to distinguish it from *M. pegasus* Alexander, and other allied members of this group. The two dististyles of the unique type are superimposed on the slide and it is possible that their conformation is not entirely as figured.

MOLOPHILUS CYGNUS sp. nov. Plate 1, fig. 22; Plate 3, fig. 48.

Belongs to the *gracilis* group and subgroup; general coloration dark grayish brown; antennæ (male) short; halteres entirely light yellow; legs dark brown; wings with a pale brown tinge, the veins and macrotrichia dark brown; male hypopygium with the dorsal and mesal lobes of basistyle blackened, the former acute at tip; both dististyles entirely blackened, simple, the outer with four or five sharp teeth before tip.

Male.—Length, about 4 millimeters; wing, 4.5.

Rostrum testaceous brown; palpi black. Antennæ (male) short, dark brown throughout; flagellar segments subcylindrical, the terminal segment a little longer than the penultimate. Head light ocherous brown in front, more grayish behind.

Anterior lateral pretergites restrictedly yellowish. Mesonotum and pleura dark grayish brown, the latter somewhat paler on the dorsal sternopleurite. Halteres uniformly light yellow, clothed with golden-yellow setæ. Legs with the coxæ and trochanters yellow; femora brown, more yellowish brown basally; tibiæ and tarsi dark brown. Wings (Plate 1, fig. 22) with a pale brown tinge, the prearcular and costal regions, together with vein Cu_1 more yellowish; veins pale brown, more yellowish as above indicated; macrotrichia dark brown, more heavily grouped along cord. Costal fringe and macrotrichia of veins long and conspicuous. Venation: R_2 lying just distad of level of r-m; petiole of cell M_3 short, about one-third to one-half longer than m-cu; vein 2d A ending about opposite m-cu.

Abdomen dark brown. Male hypopygium (Plate 3, fig. 48) with the dorsal lobe of basistyle, *db*, slender, the extreme tip

acute and heavily sclerotized; remainder of lobe with coarse setæ; mesal lobe slender, heavily blackened, appearing as a nearly straight rod, the apex rounded; ventral lobe, *vb*, large, spatulate, with erect to feebly retrorse setæ. Two dististyles, both simple, entirely blackened and strongly curved or sinuous, the outer, *od*, broader and provided with four or five sharp teeth immediately before the acute tip.

Habitat.—China (Szechwan).

Holotype, male, Kwanhsien, altitude 4,500 feet, August 13, 1930 (*Franck*).

Molophilus cygnus belongs to the section of the subgroup having short antennæ in the male sex, uniformly pale halteres, with the mesal lobe of the basistyle of hypopygium slender and heavily blackened, and with the dorsal lobe of basistyle acutely pointed at apex. The nearest regional allies seem to be the Formosan *M. arisanus* Alexander and *M. issikii* Alexander, in which the conformation of the mesal lobe of the basistyle and of both dististyles is entirely distinct.

ILLUSTRATIONS

[*a*, ædeagus; *b*, basistyle; *d*, dististyle; *db*, dorsal lobe of basistyle; *g*, gonapophysis; *ia*, inner arm of inner dististyle; *id*, inner dististyle; *oa*, outer arm of inner dististyle; *od*, outer dististyle; *s*, sternite; *t*, tergite; *vb*, ventral lobe of basistyle; *vd*, ventral dististyle.]

Plate 1

Fig. 1. *Dolichopeza* (*Nesopeza*) *francki* sp. nov., venation.
2. *Tipula seticellula* sp. nov., venation.
3. *Tipula* (*Tipula*) *okinawensis* sp. nov., venation.
4. *Liogma brevipecten* sp. nov., venation.
5. *Liogma fuscipennis* sp. nov., venation.
6. *Limonia* (*Dicranomyia*) *trifilamentosa* sp. nov., venation.
7. *Limonia* (*Idioglochina*) *tokunagai* sp. nov., venation.
8. *Proantocha quadrivittata* sp. nov., venation.
9. *Antocha sagana* sp. nov., venation.
10. *Dicranoptycha machidana* sp. nov., venation.
11. *Helius* (*Helius*) *pluto* sp. nov., venation.
12. *Polymera parvicornis* sp. nov., venation.
13. *Limnophila* (*Elæophila*) *serrulata* sp. nov., venation.
14. *Eriocera nigrotrochanterata* sp. nov., venation.
15. *Eriocera kelloggi* sp. nov., venation.
16. *Eriocera kamiyai* sp. nov., venation.
17. *Atarba* (*Atarbodes*) *bipunctulata* sp. nov., venation.
18. *Gonomyia* (*Gonomyia*) *subcognatella* sp. nov., venation.
19. *Ormosia tokunagai* sp. nov., venation.
20. *Erioptera* (*Ilisia*) *subareolata* sp. nov., venation.
21. *Molophilus albireo* sp. nov., venation.
22. *Molophilus cygnus* sp. nov., venation.

Plate 2

Fig. 23. *Dolichopeza* (*Nesopeza*) *francki* sp. nov., male hypopygium.
24. *Tipula seticellula* sp. nov., male hypopygium, lateral.
25. *Tipula seticellula* sp. nov., male hypopygium, ninth tergite.
26. *Tipula seticellula* sp. nov., male hypopygium, dististyles.
27. *Tipula seticellula* sp. nov., male hypopygium, gonapophysis.
28. *Tipula seticellula* sp. nov., male hypopygium, eighth sternite.
29. *Tipula* (*Tipula*) *okinawensis* sp. nov., male hypopygium, details.
30. *Tipula* (*Vestiplex*) *divisotergata* sp. nov., male hypopygium, details.
31. *Liogma brevipecten* sp. nov., antenna, basal six flagellar segments.
32. *Liogma fuscipennis* sp. nov., antenna, basal five flagellar segments.
33. *Limonia* (*Dicranomyia*) *trifilamentosa* sp. nov., male hypopygium.
34. *Limonia* (*Idioglochina*) *tokunagai* sp. nov., male hypopygium.
35. *Antocha sagana* sp. nov., male hypopygium.

PLATE 3

FIG. 36. *Dicranoptycha machidana* sp. nov., male hypopygium.
37. *Helius* (*Helius*) *pluto* sp. nov., male hypopygium.
38. *Polymera parvicornis* sp. nov., male hypopygium.
39. *Limnophila* (*Elæophila*) *serrulata* sp. nov., male hypopygium.
40. *Limnophila imanishii* sp. nov., antenna, female.
41. *Atarba* (*Atarbodes*) *bipunctulata* sp. nov., male hypopygium.
42. *Chionea nipponica* sp. nov., male hypopygium.
43. *Chionea araneoides* Dalman, male hypopygium.
44. *Gonomyia* (*Gonomyia*) *subcognatella* sp. nov., male hypopygium.
45. *Ormosia tokunagai* sp. nov., male hypopygium.
46. *Erioptera* (*Ilisia*) *subareolata* sp. nov., male hypopygium.
47. *Molophilus albireo* sp. nov., male hypopygium.
48. *Molophilus cygnus* sp. nov., male hypopygium.

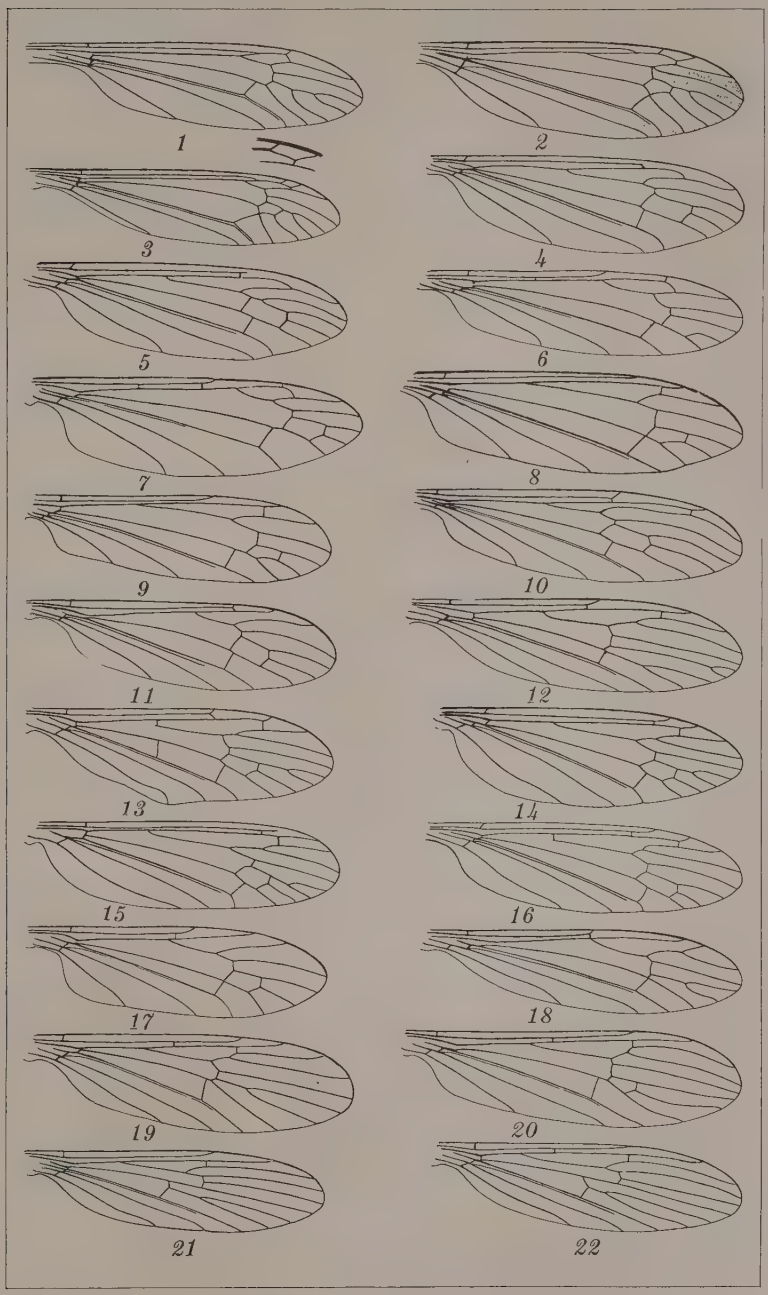

PLATE 1.

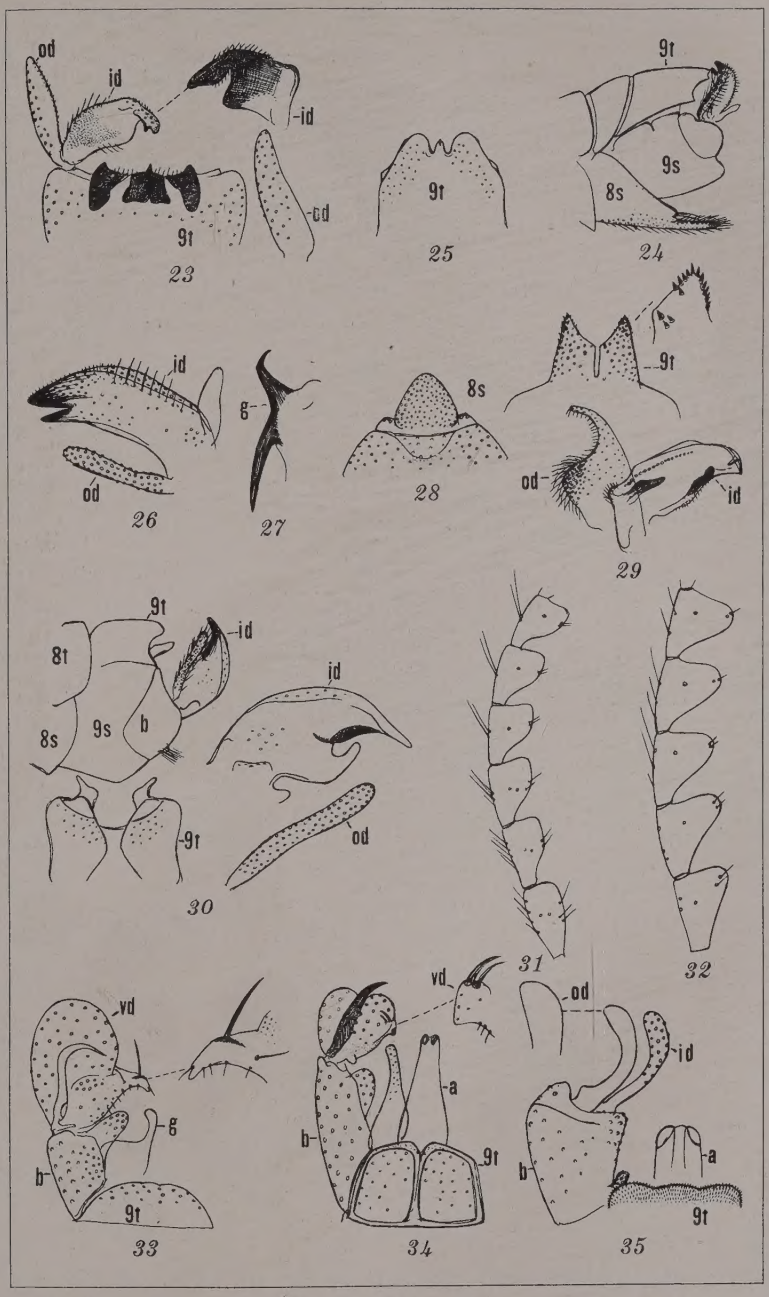
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PLATE 2.

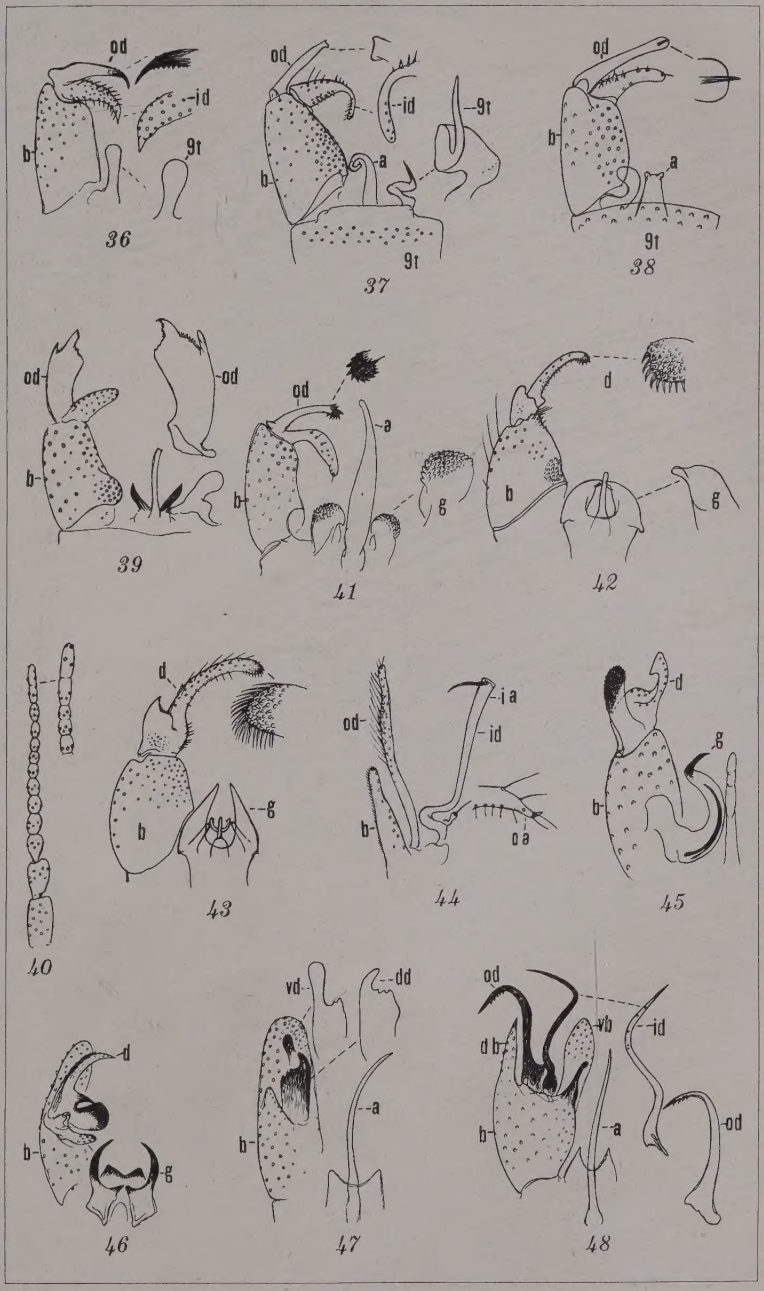
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PLATE 3.